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LIMITATIONS OF THE ELECTROCARDIOGRAM AS AN AID IN THE DIAGNOSIS OF CORONARY OCCLUSION*

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THE study of the electrocardiogram in cases of coronary occlusion has shown that there are certain changes frequently associated with this clinical condition. The changes which have been generally accepted as corroborative or suggestive of the clinical diagnosis are the following: (1) a high take-off and a peculiar rounding of the T wave as described by Pardee[1], (2) low amplitude of the ventricular complexes; (3) a peculiar V-shaped inversion of the T wave occurring as a later development. Other changes in the electrocardiogram which may be seen in cases of coronary thrombosis, but which are less characteristic because they are so common in other conditions are spreading of the Q-R-S complex with or without notching, alteration in the A-V conduction, and the presence or development of a prominent Q-3, a feature noted by Levine and Brown[2] and Pardee[3], the significance of which is not clearly understood. A most striking characteristic, however, is the marked variability of the tracings from day to day. On account of this variability it sometimes happens in cases presenting the clinical picture or history of coronary occlusion, that normal tracings are obtained, or at least significant changes are lacking at certain periods during the course of the disease. During the past several years there has been a good deal of attention directed at the changes in the electrocardiograms which suggest myocardial infarction and coronary artery disease. There have been very few data, however, of a negative type, i.e., to what extent can normal tracings be helpful in deciding that the coronary arteries are not particularly diseased? Can electrocardiography be used to rule out the diagnosis of coronary occlusion? It is this aspect of the problem with which this study is concerned‡.

Three hundred and twenty-eight cases of coronary occlusion were reviewed. Only those were studied in which electrocardiograms were taken some time after the onset of the attack. Of these, two hundred and ninety-three cases showed distinct abnormalities in the curves of one type or another. There were thirty-five in which the electrocardiogram was regarded as normal in all respects, or at least as lacking the significant alterations mentioned above. There were no cases included in the latter group in which the clinical diagnosis was at all doubtful. These latter thirty-five cases served as the primary object of our investigation. Realizing that the character of the curves was likely to change from day to day and from week to week, the exact time that normal curves were found became a matter of considerable importance.

There are in general two points of view with regard to the diagnostic aid that the electrocardiogram might render—first, as an aid in the diagnosis during the acute stages of the disease (days or weeks), and, secondly, in the diagnosis of an attack that occurred months or years previously. How important is a normal tracing in deciding that the present illness is not due to an acute coronary occlusion? Furthermore, how important is the finding of normal tracings in deciding that a patient never had an attack of coronary occlusion in the past?

It was found that the thirty-five "normal" cases could be divided into three groups. In the first group were included seven cases in which a normal electrocardiogram was obtained within the first few weeks following the attack, while subsequent tracings presented characteristic changes. In the second group there were nineteen cases in which the first electrocardiograms showed characteristic changes which subsequently were replaced by normal tracings. The remaining nine cases had either only one electrocardiogram which was normal, or several tracings none of which presented any of the characteristic changes.

As an illustration of the progressive changes in the electrocardiogram that can occur in succession days and weeks following an attack, the records of a case are reproduced in Fig. 1. The very marked changes here occurred in leads two and three. In other cases similar changes take place in lead one, with a resultant inversion of T-1 and upright T-3. In Figures 2 to 12 are reproduced some of the electrocardiograms of

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‡Recently an analysis was made of the appearance time of the T wave changes in coronary thrombosis by Hurxthal (*Arch. Int. Med.*, 1930, XLVI, 657), but the point of view emphasized in this study was not taken up.

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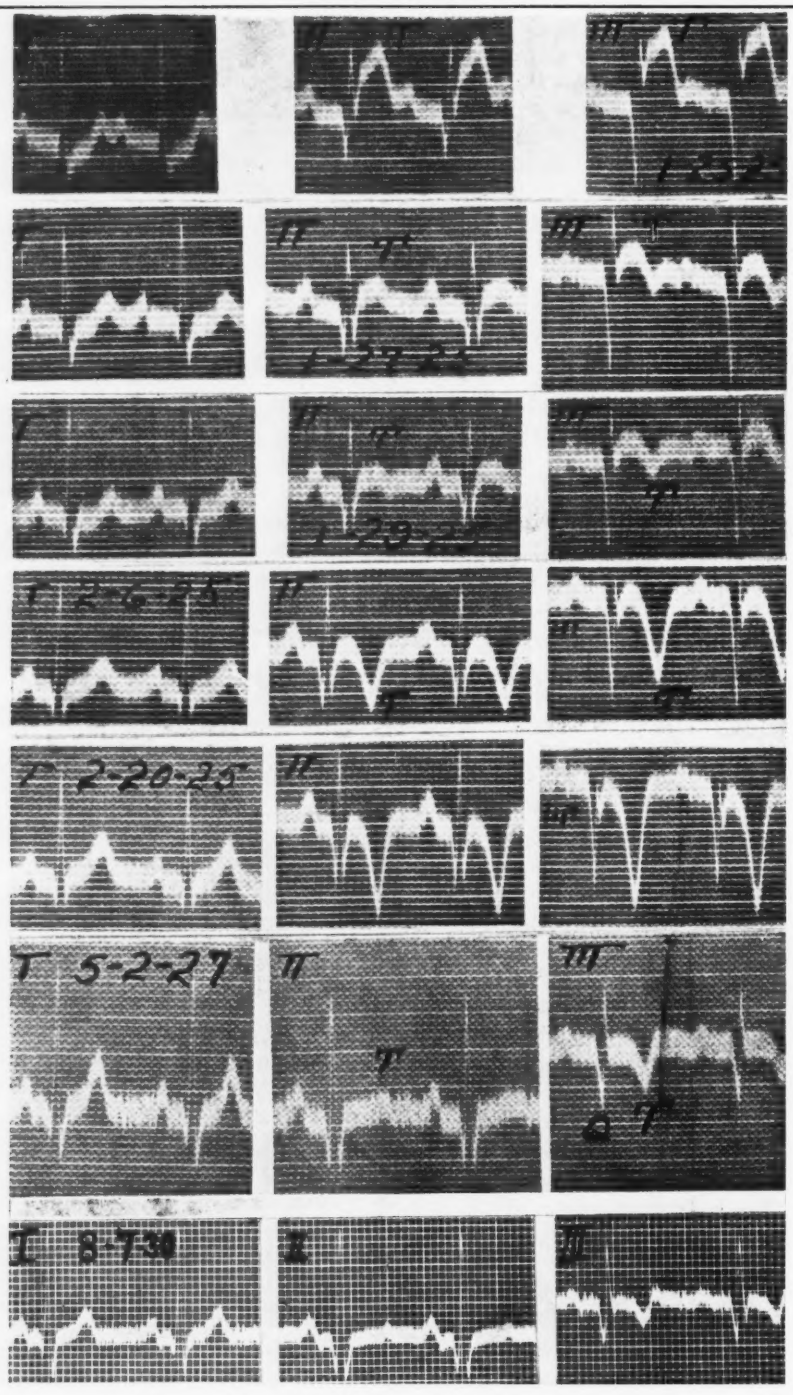

FIG. 1. This series of tracings illustrates the rapid changes that take place in the form of the electrocardiograms. During the last three years the curves were essentially unchanged.

cases in this study which showed normal curves at one time or another. In some the normal tracing is followed by significant changes while in others the reverse takes place. From Fig. 2 it is quite clear that curves may be obtained so early after the onset of the attack that nothing remarkable may be detected, whereas some days later the electrocardiogram may become more diagnostic. The same is true of Figs. 3 to 9. In Fig. 6 a slight change in T-1 in the first tracing became more marked the following day, and two days later had entirely disappeared. In this case it is apparent that if the tracings were obtained only on July 15, 1927, i.e., five days after the onset of the attack, it would have been necessary to regard them as normal. In this way it can readily be seen that patients probably have had attacks of coronary occlusion with temporary alterations in the electrocardiograms who subsequently have normal curves. In Figs.

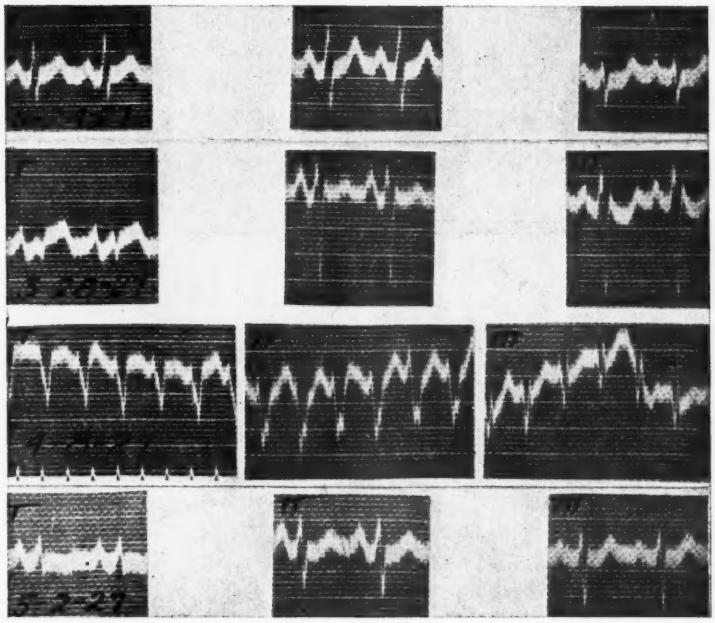

FIG. 2. First tracings were obtained five hours after onset of attack and can be regarded as essentially normal. The next day (tracings not included) and subsequently distinct alterations of the ventricular complexes were noted. On April 8, 1927 paroxysmal ventricular tachycardia was present.

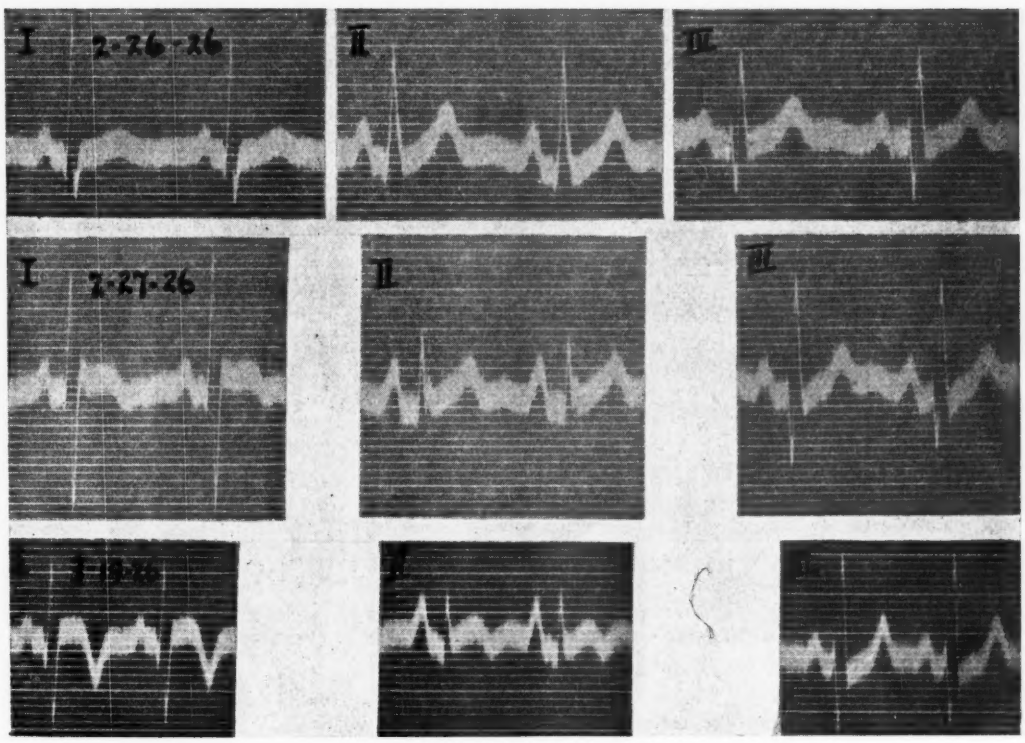

FIG. 3. First tracings were obtained twelve hours after the onset of the attack. Although a definite Q-3 was present and there was practically no iso-electric R-T interval, none of the more striking changes in the complexes had as yet occurred. Note the subsequent alterations in the T wave in Leads 1 and 2.

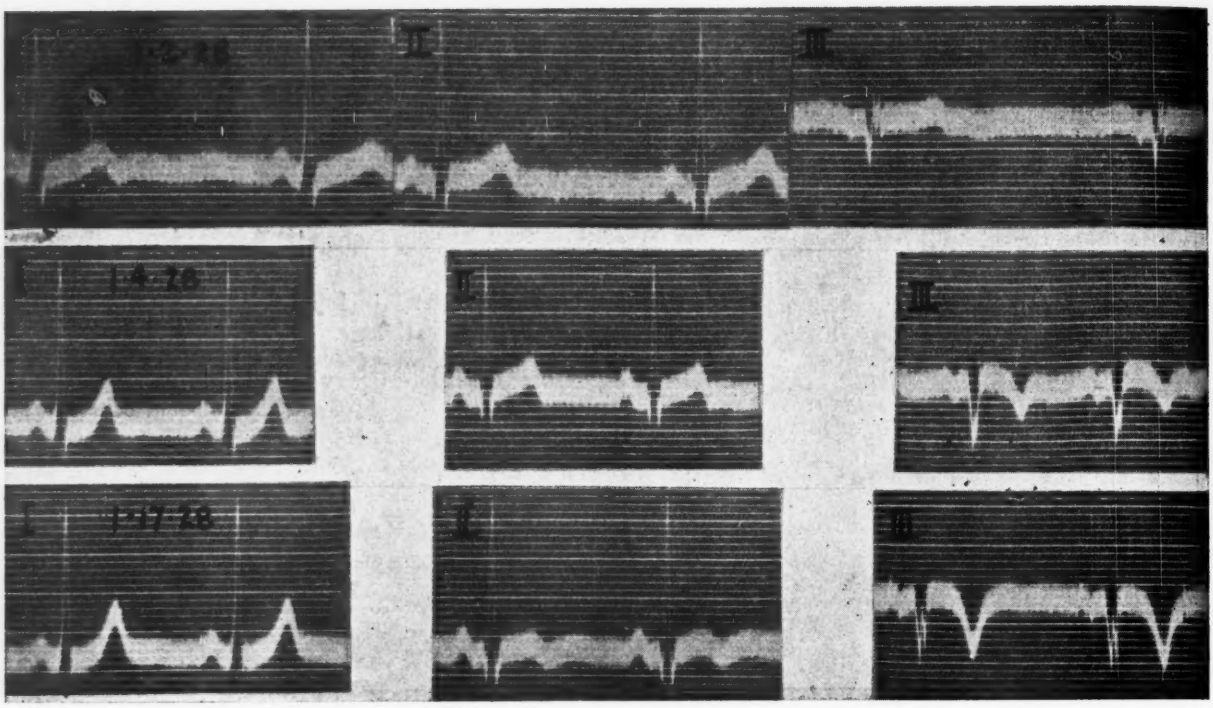

FIG. 4. First tracings taken forty-eight hours after onset of attack. No striking changes were noted, although two days later T-3 was definitely dipped.

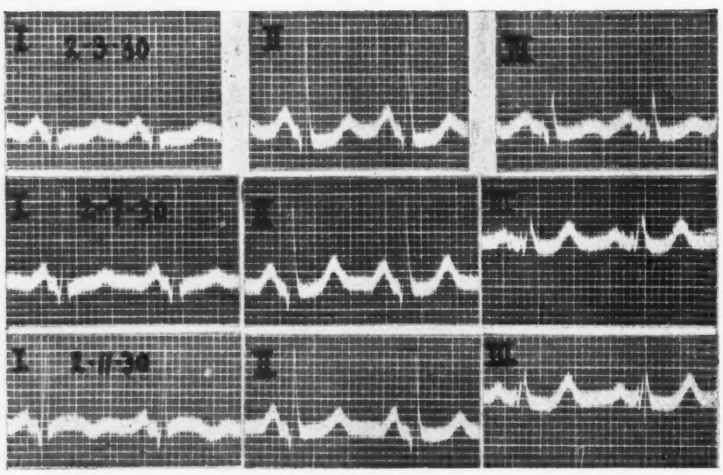

FIG. 5. Attack occurred January 31, 1920. Nothing remarkable in the tracings for seven days after attack. Curves taken February 11, 1930 showed fairly typical rounding of T-1.

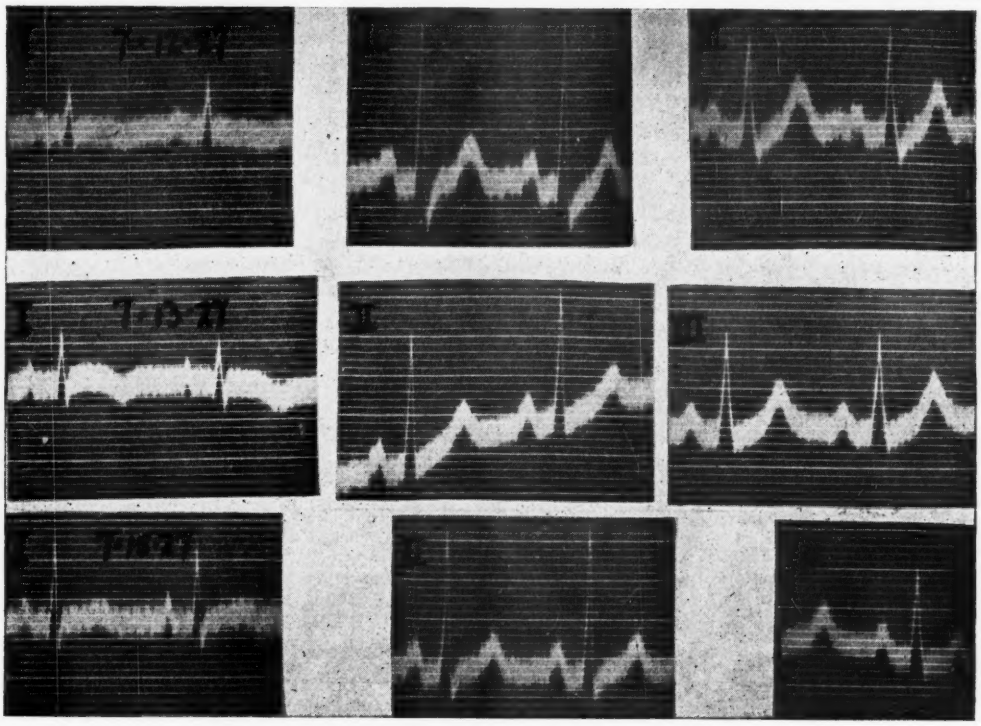

FIG. 6. Attack July 11, 1927. Siginficant changes in T-1 on July 13, 1927. Two days later this change had practically disappeared, so that on July 15, 1927 the curves might properly be regarded as normal.

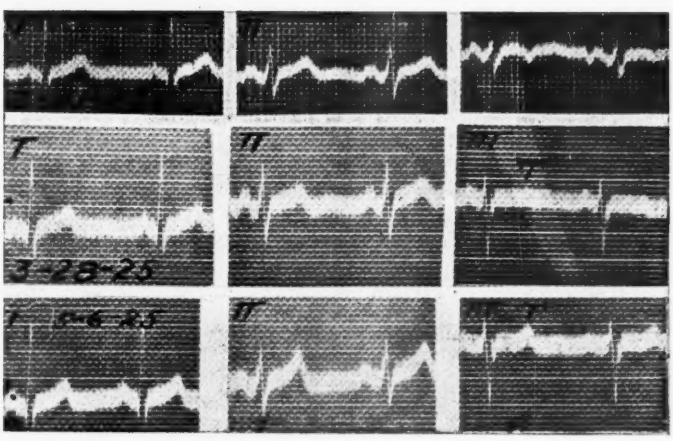

FIG. 7. Attack March 13, 1925. Eight days later T-3 was typically distorted. This gradually returned to normal in about two months. The last tracing showed normal upright T waves in all leads.

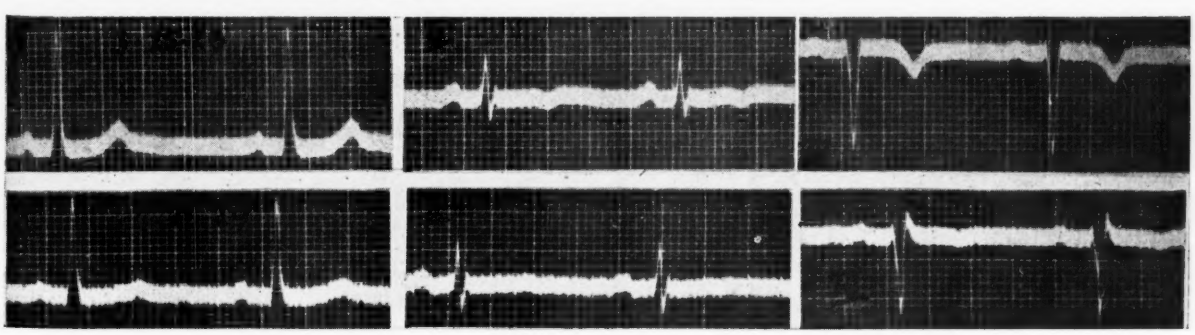

FIG. 8. Attack March 4, 1929. First tracings taken March 25, 1929. Note sharply inverted T-3. This had entirely disappeared when the next tracing was taken on July 25, 1929.

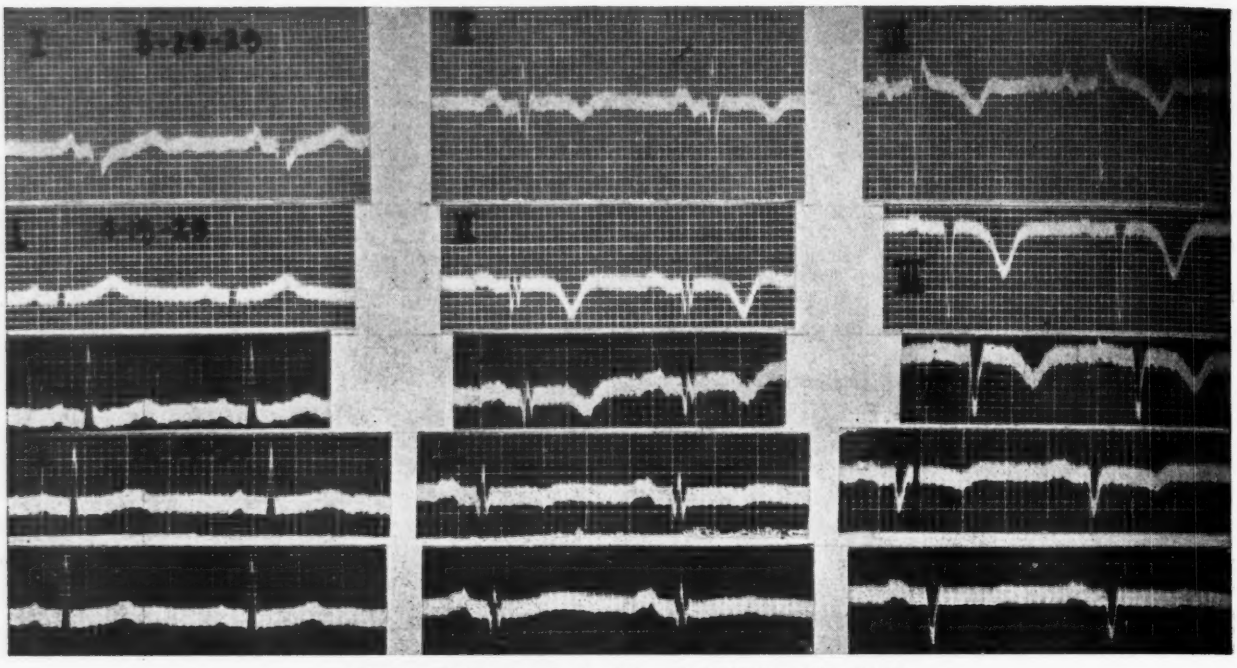

FIG. 9. Attack March 28, 1929. First tracing taken March 29, 1929 showed changes in T-2 and T-3. These became more prominent during the next two weeks. The last tracing taken sixteen months later (August 4, 1930) merely showed flat T waves.

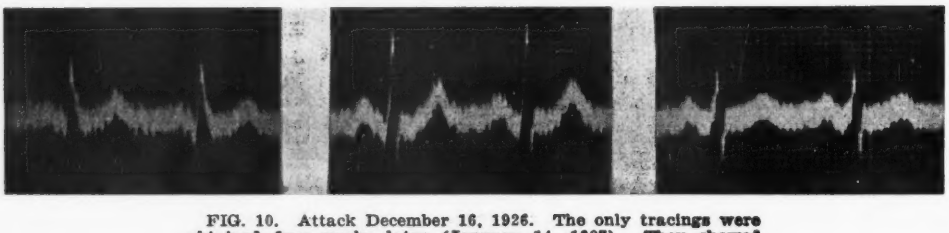

FIG. 10. Attack December 16, 1926. The only tracings were obtained four weeks later (January 14, 1927). They showed no important variations from normal.

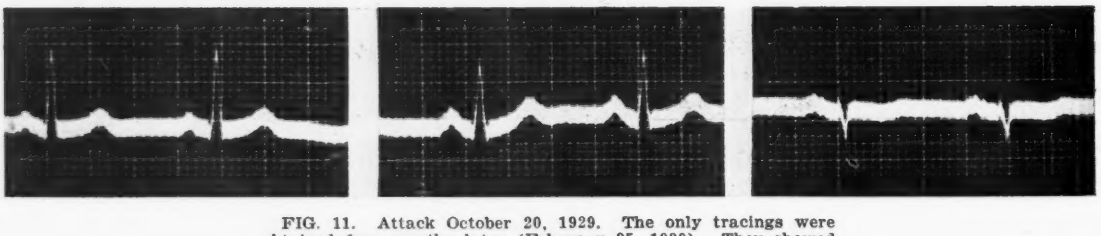

FIG. 11. Attack October 20, 1929. The only tracings were obtained four months later (February 25, 1930). They showed no important variations from normal.

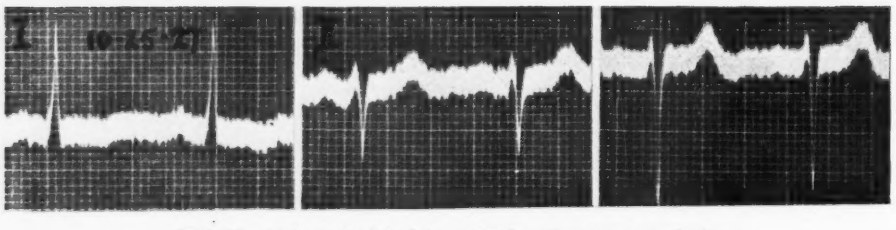

FIG. 12. This patient had two attacks of coronary occlusion, the first in July, 1925, the second in July, 1927. The only tracing was obtained October 25, 1927. It showed merely a flat R-T interval.

10 to 12 the only tracings that were taken showed none of the abnormalities characteristic of coronary occlusion. No doubt had other electrocardiograms been made during the early days after the onset of the attack, changes similar to those shown in Figs. 1 to 9 would have been found.

Of particular interest are Figs. 5, 6, 7, and 10. Here the "normal" electrocardiograms do not show even the vague suspicious evidences of coronary occlusion. The T waves are all upright and Q-3 waves are absent. Three of them do not even show any left ventricular preponderance which is otherwise so common in coronary artery disease. In some of the other figures that were considered essentially normal, using the eye of faith, one can detect very minor alterations that no doubt were real, as subsequent tracings showed further changes of the same character. In Fig. 3 the T wave in lead two of the first tracing starts to rise directly with the descent of the T wave, leaving no iso-electric interval. The final tracing shows a more characteristic change in T-2. Also in Fig. 4 the R-T interval in lead three of the first tracing really lies on a slightly higher level than the iso-electric line. Here again that same T wave becomes more characteristic in subsequent curves. It is therefore clear that many electrocardiograms which individually one has to consider essentially normal really contain slight but significant changes that only become apparent if compared with tracings taken at other times.

In general there are two periods during which essentially normal curves occurred. On the one hand normal tracings were found during the first few days after an attack, the characteristic changes occurring later. Contrariwise, curves may return to normal after early alteration has occurred. There were only eight cases in the entire series of three hundred and twenty-eight cases that showed normal curves during the first ten days. The return to normal in those that previously showed significant alterations in the electrocardiograms took place with very varying speed. In one case the changes disappeared four days after the onset (Fig. 6), whereas in another slight changes which finally disappeared were still present seven months after the attack (Fig. 9). It must be clear that, considering the large number of cases reviewed in this study, the finding of normal electrocardiograms in a case of coronary thrombosis is comparatively rare and would be still rarer if more frequent tracings were taken during the acute stage. However, because it does occur, it cannot be used to exclude the diagnosis of coronary occlusion.

SUMMARY

1. In a study of 328 cases of coronary occlusion in which the electrocardiograms were taken, there were 35 that showed essentially normal tracings at one time or another following the onset of attack.

2. For the purpose of this study, curves were regarded as essentially normal if they lacked the following changes; alterations in the T-wave that are generally regarded as occurring with acute coronary occlusion (high take-off, convexity and sharp dipping of the T wave), low amplitude, disturbances of A-V conduction, and spreading of the Q-R-S complex.

3. Because tracings were not taken with the same frequency or at fixed time intervals, it was impossible to draw any statistical conclusions, except of a very vague nature. In most cases several curves were made in the first week or two; in a small number the only ones obtained were made months after the attack.

4. In six cases, so-called normal electrocardiograms were obtained from a few hours to seven days after the onset of attack. In all these, subsequent tracings showed characteristic alterations in the ventricular complexes.

5. In twenty cases typical changes in the tracings taken soon after the onset of the attack were replaced by "normal" curves at some subsequent time. The return to normal varied greatly, coming as quickly as four days after the onset of attack in one case and more gradually and requiring several months in another.

6. In nine cases the only electrocardiograms obtained were normal. In only one of these nine cases were curves obtained within the first ten days.

7. Of the entire series of 328 cases there were only eight in which "normal" electrocardiograms were obtained at any time during the first ten days.

8. Regardless of the time elapsing after an attack, a normal electrocardiogram in a case considered clinically as coronary occlusion does not exclude the diagnosis.

9. A most important feature in this problem is the marked changes in the electrocardiogram that occur from day to day especially during the first two weeks. By taking repeated tracings therefore, normal curves may be found later to change to those more characteristic of coronary artery disease.

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CHRONIC ULCERATIVE COLITIS*

BY EVERETT D. KIEFER, M.D.†

FORTUNATELY, chronic ulcerative colitis is not a common disease, but, on the other hand, it is not a rare disorder, for in a period of somewhat less than four years twenty-five cases have been seen at the Lahey Clinic. It may vary in severity between wide limits; it may be a fulminating, rapidly fatal disease, or only a comparatively mild diarrhea, which the patient may have for years without confining him to bed. (Table No. 1.) There were seven patients in our group who had a severe bloody diarrhea, septic fever, toxemia, and severe anemia; five of these patients died in the hospital. Twelve, or about half the patients, had a somewhat less severe bloody diarrhea with little or no fever and toxemia. The remaining six were comparatively mild, ambulatory cases.

TABLE NO. 1

CHRONIC ULCERATIVE COLITIS

Report based on experiences with 25 cases seen at the Lahey Clinic in four years

SYMPTOMATOLOGY

Severe (7 cases)

1. Severe bloody diarrhea.
2. Septic fever.
3. Toxemia.
4. Severe anemia.
5. Death (5 cases).

Moderately Severe (12 cases)

1. Bloody diarrhea.
2. Mild or no fever.
3. Moderate anemia.

Mild (6 cases)

1. Frequent loose stools.
2. Small amount of blood in stools.
3. Mild anemia.

TABLE NO. 2

DIAGNOSIS

Proctoscopic Examination

1. Diagnostic in 88 per cent. of cases.
2. Mucosa is edematous and covered with exudate. Surface is rough, granular and shows numerous small bleeding points.
3. Exudate consists of mucus, pus, blood and bacteria.
4. Polypi (1 case).
5. Carcinomatous degeneration (1 case).

X-Ray Examination

1. Marked irritation.
2. Loss of haustrations.
3. Loss of flexibility.
4. Atresia of colon or rectum.

In making the diagnosis proctoscopic examination is the most important procedure. (Table No. 2.) It is absolutely diagnostic in a high percentage of cases. In our group there were only three cases in which the disease was localized to a portion of the colon above the reach of the sigmoidoscope. The proctoscopic findings are characteristic. The edema of the intestinal wall gives to the examiner the impression of looking into a rigid thick-walled tube, which is frequently not collapsed but open so that considerable distance can be seen beyond the end of the instrument. The mucosa is covered with a pinkish-gray exudate consisting of mucus, blood, pus and bacteria. When this is wiped away, there is disclosed a roughened, granular surface, which is dark red and bleeds freely from many small points. The process involves the entire mucosa, there being no areas of normal mucous membrane. The proctoscope may also disclose polypi and malignant degeneration, each of which occurred once in our series.

TABLE NO. 3

ETIOLOGICAL FACTORS

Bacterial Infection

1. Bargen's diplococcus not yet generally accepted.
2. Many organisms present. Difficulty in distinguishing secondary invaders from primary causative organisms.

Avitaminosis

1. Advanced as a cause.
2. Probably frequently present due to dietary restrictions.

Allergic and Nervous Disorders

1. Theory has few supporters.

Intestinal Amebiasis

1. Ameba histolytica found in stools of 16 per cent. of our cases.
2. Craig complement fixation test positive in 15 cases (79 per cent.) out of 19.
3. Chronic cases of amebic dysentery become secondarily infected, which modifies the type of ulceration and makes it difficult to find amebae in the stools or to make a differential diagnosis with the proctoscope.

The x-ray is not always diagnostic, but shows marked irritation of the intestine as well as loss of haustrations. Figure 1 shows by means of a barium enema the characteristic smooth, tube-like colon and, after evacuation of the enema, the exaggerated emptying of a markedly irritable bowel. Long-standing cases produce the so-called "lead pipe" colon, in which the wall has become so infiltrated that it is rigid and cannot contract when the bowel is emptied.

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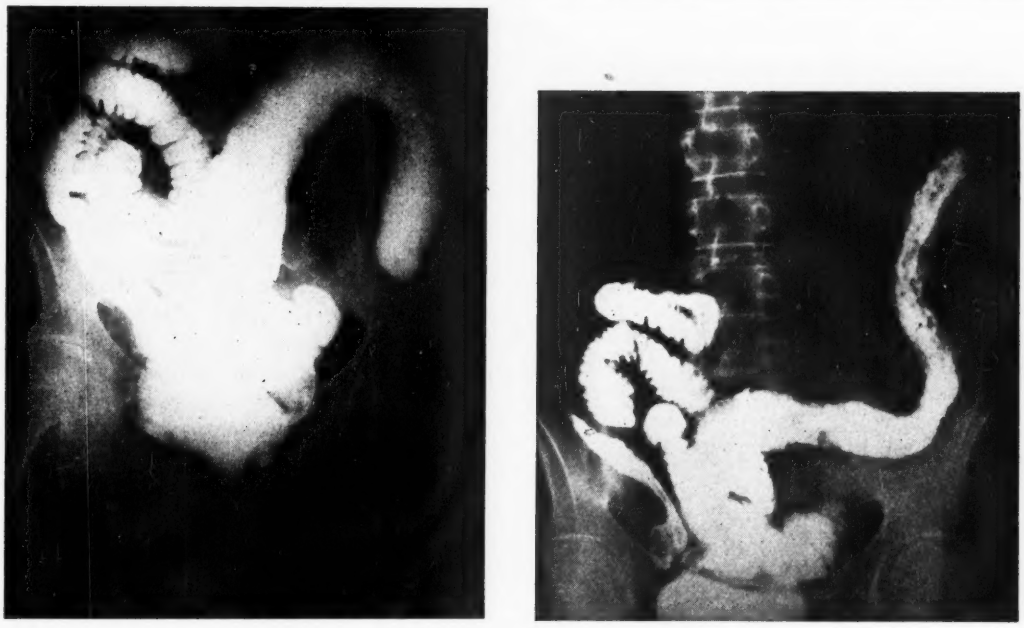

FIGURE 1. The first film shows a barium enema which demonstrates the smooth, tubelike colon with practically no haustrations. It does not hold the enema solution well and the upper colon has already emptied itself, forcing the barium into the dilated sigmoid. It also shows a patent ileocecal valve which allows the filling of the dilated loops of terminal ileum. The second film, taken after evacuation of the enema, shows the ascending and transverse colon contracted to a stringlike appearance. The exaggerated emptying indicates marked irritation, but no loss of flexibility of the bowel.

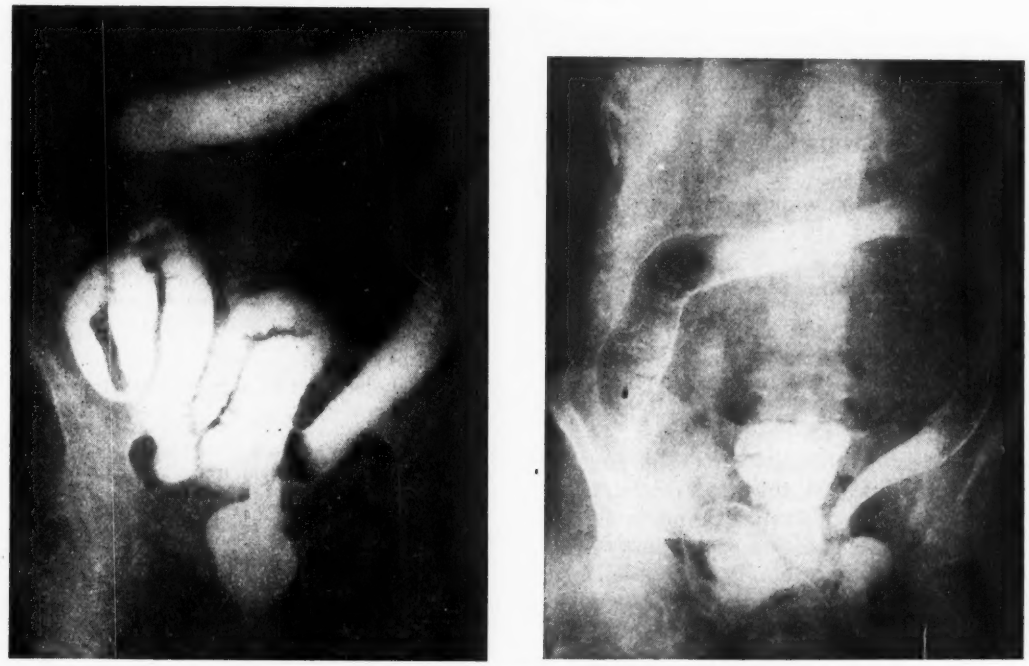

FIGURE 2. These roentgenograms show a barium enema in a long-standing case of ulcerative colitis.
There is marked rigidity and loss of flexibility so that after evacuation of the enema there is some shortening of the bowel, but no diminution of the caliber. This is sometimes called the "lead pipe" colon.

(Figure 2.) Cicatricial contractions following the healing of ulcerations may result in stenosis or atresia of the colon or rectum.

The etiology (Table No. 3) of the disease is very much unsettled and disputed. Bargen's[1] work has greatly stimulated the study of the bacteriology of ulcerative colitis, but the diplococcus which he described has not yet been

generally accepted as the cause of the disease. The chief obstacle in the bacteriological study appears to be the difficulty in distinguishing between secondary invaders and primary causative organisms. Deficiency of vitamines in the diet has been advanced as a cause. This condition is frequently present in ulcerative colitis because of long-standing dietary restrictions and is an important consideration in treatment. The theories that allergic and nervous disorders are causative factors have few supporters.

The stools of all our patients have been searched carefully for pathogenic amebae and we have found the end ameba histolytica in 16%. The complement fixation for infection with this ameba, a test which has been developed by Craig[2] and which in his hands has been diagnostic in a high percentage of cases, was strongly positive in fifteen out of nineteen cases. This evidence (recently published by the author[3]) makes it seem possible that an initial amebic infection may be more than a coincidence. Patients with chronic amebic dysentery become secondarily infected, a condition which modifies the type of ulceration and makes it difficult either to find the ameba in the stools or to make a differential diagnosis with the proctoscope.

TABLE NO. 4

TREATMENT

Diet

1. High calorie.
2. Low residue.
3. High vitamine—orange juice, yeast, cod liver oil.
4. High iron—liver.

Drugs

1. Anti-amebic.
   a. Emetine hydrochloride — intramuscularly.
   b. Acetarsone (Stovarsol) arsenical — by mouth.
   c. Bismuth — by mouth.
   d. Yatren — rectal irrigations.
2. Anti-spasmodic.
   a. Belladonna.
   b. Calcium and parathormone.
3. Blood forming.
   a. Ferric ammonium citrate.

Physiotherapy

1. Ultraviolet light.
2. Heat—external and hot drinks.

Surgery

1. Transfusion.
2. Ileostomy.

In the treatment the diet should be high in calories to maintain nutrition, low in residue to rest the colon, high in vitamines to overcome previous dietary deficiencies and high in iron to support blood restoration. (Table No. 4.)

In our opinion, a course of antiamebic drugs should be given in all cases regardless of whether or not the amebae are found in the stools. Emetine intramuscularly is toxic for the amebae buried within the tissues. Acetarsone kills those within the lumen of the intestine. Bismuth also is toxic for amebae as well as soothing for the ulcerated intestinal wall. Yatren is useful as a rectal irrigation, but on the whole irrigations of any kind are not well tolerated and have not been used in our cases to much extent. Belladonna and calcium combined with parathormone are useful antispasmodics.

It is important to treat the marked secondary anemia which is usually present by giving large doses of iron salts. Ultraviolet light is an adjunct of treatment and heat in the form of hot drinks or a hot water bottle on the abdomen has a soothing antispasmodic effect. Blood transfusion is extremely valuable and should be used frequently to replace a rather considerable blood loss.

Ileostomy is the only operation which puts the colon at complete rest and this is the sole object of surgical treatment. Although appendicostomy and cecostomy facilitate irrigations of the bowel and have the added advantage of being easily closed, they do not completely divert the fecal stream so that the more important factor of complete rest for the colon is not obtained. It has been our experience that irrigations with any solution are of questionable value and if they were desirable, it appears that almost as satisfactory irrigations could be carried out from below as from above through openings in the appendix or cecum. An ileostomy (Table No. 5) is extremely important as a life-saving procedure for patients with severe toxemia, who are responding poorly to medical measures. The clinical improvement which follows the operation is sometimes miraculous. Excellent clinical judgment is necessary to decide when the operation is indicated; the greatest danger lies in delaying until it is too late to save the patient. On the other hand, one is faced with an almost

TABLE NO. 5

SURGICAL TREATMENT (ILEOSTOMY)

Indications

1. Severe toxemia.
2. Poor response to medical measures.
3. Atresia of colon or rectum.

Danger in Delay

1. Tendency is to put off operation until too late.

End Results

1. Permanent small intestinal fistula.
2. Progressive destruction of the colon and rectum.

Clinical Results in 8 Cases

1. Postoperative death: 4 cases.
2. Repeated recurrences: 1 case.
3. Well but with bloody rectal discharge: 2 cases.
4. Well but with atresia of rectum: 1 case.

certain permanent small intestinal fistula, which is somewhat more trying than a colostomy. Lahey has frequently stated that if the disease is severe enough to make an ileostomy necessary, the colon will become so completely destroyed that it can rarely be used again.

Of eight patients who have had ileostomies, four died postoperatively. Of the other four, one has had recurrences of typical attacks of the disease, two still have bloody rectal discharges and one developed almost complete occlusion of the rectum. Our experiences in the last four cases indicate that although the more severe general effects of the disease may be relieved by putting the colon at rest, the local destruction of the colon may proceed unabated. In moderately severe and mild cases an ileostomy is never indicated except when late lesions cause obstruction in the rectum or colon.

Fourteen patients have been treated medically, including one or more courses of antiamebic drugs. (Table No. 6.) Nine patients have had excellent remissions and four of these have remained well for over a year. Four cases have been improved and one has not received much lasting benefit. Two more patients have just been discharged from the hospital apparently in a good remission.

TABLE NO. 6

MEDICAL TREATMENT INCLUDING ANTI-AMEBIC DRUGS

Clinical Results in 14 Cases

1. Excellent remission for over one year: 4 cases (1 severe) (3 mod. severe).
2. Excellent remission, followed less than 1 year: 5 cases (1 severe) (2 mod.) (2 mild).
3. Improved: 4 cases (1 severe) (1 mod.) (2 mild).
4. Recurrent attacks: 1 case (mod.).

Although it is decidedly unsafe to draw conclusions from therapeutic results in a disease such as ulcerative colitis, the apparent good effects of antiamebic treatment tend to support the importance of amebic infections in these cases.

CONCLUSIONS

Experiences with twenty-five cases of chronic ulcerative colitis have been reported.

There were seven very severe cases, in five of which the patient died.

The proctoscopic and sigmoidoscopic examination is the most important diagnostic procedure.

The etiology is unsettled, but the possible importance of amebic infections is discussed.

Treatment consists of a suitable diet, antiamebic drugs, antispasmodics, supportive measures, treatment for the secondary anemia, blood transfusions and ileostomy.

Ileostomy is the operation of choice because it puts the bowel at complete rest.

The operation is important as a life-saving measure in severe cases, but does not stop the local destruction in the intestinal wall.

The therapeutic results with antiamebic treatment have been sufficiently favorable to lend importance to the possibility of a primary amebic infection.

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MORTALITY AND EXPECTATION OF LIFE IN MENTAL DEFICIENCY IN MASSACHUSETTS: ANALYSIS OF THE FOURTEEN-YEAR PERIOD 1917-1930*

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INTRODUCTION

IN the past there has been a great deal of comment and conjecture upon death rates among the feebleminded. The mortality appeared to be higher than that of the general population, but little or no exploration was undertaken concerning the details of this important incidence. The few investigations which have delved into the problems have been rather general in character, treating the subject of feeblemindedness as a whole instead of weighing the relative importance of each of its subdivisions, the mental status groups idiots, imbeciles and morons.

We observe, in addition, that a proper value was not assigned to the matter of age. As is well known, our State school populations are comprised almost entirely of the younger age groups and cluster about a median of twenty years. Approximately 95 per cent. are under the age of forty. The U. S. Census Report[1] on feebleminded and epileptics in institutions states that 65.7 per cent. are between the ages of ten and thirty years, and that only 1 per

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cent. are above the age of sixty*. In spite of this we have, in the past, compared the gross death rates of these institution populations with the death rates of the general population of the State or country. The latter rates, however, cover the entire life span and, for our purposes, are loaded with higher rates in the upper age groups. Thus, in comparing the two groups, we have been confronted with a death rate for the feebleminded which was two or three times that of the general population. We know that this cannot be exact. However, it may be well at this point to review briefly the material which is available on the subject.

Tredgold[2] in his study of 2,219 consecutive deaths in two English institutions, emphasized the fact that "in the mentally deficient the highest death rate falls between the tenth and twentieth years, the period of life in which the mortality rate is lowest among the general population†." However, Tredgold's figures actually showed that the largest *number* of deaths occurred between the ages of ten and twenty years, and did not show anything whatever in reference to death *rates*.

Another important omission in our present knowledge exists in the information on death rates for the mental status groups idiots, imbeciles and morons. Tredgold believed that the mortality had a direct relation to the *degree* of deficiency, although he had no specific data on the subject. Lapage[3] came to the conclusion that "mentally defective persons die at a lower age than is normal, and although this is probably more marked in idiots and imbeciles, the term of life of feebleminded persons is less than average‡." Again, Barr[4], in studying the death periods and causes in 625 cases, came to the conclusion that few feebleminded children pass the 25th year, and most die between ten and twenty years§.

The U. S. Census Report[1] on feebleminded and epileptics in institutions gives us the only material to date on death rates in the mental status groups. It is as follows: "The death rate among the feebleminded varies in accordance with the degree of mental defect, the general average rates among idiots, imbeciles and morons per 1,000 under treatment being 68.7, 26.7 and 10.1 respectively. The death rate among males in each mental status group is higher than among females. It is noteworthy that this death rate in the moron group approximates the death rate of the general population of the country as a whole‖." This brings out the very significant fact that there is a decided difference in the number of deaths within each grade of mental defectiveness, and that imbeciles have a death rate comparatively two and a half times that of morons, while the idiots have a death rate nearly seven times that of the morons.

Again, we have little other than general facts regarding the *causes* of death among mentally defective children. We have not studied, except in a general way, their susceptibility to the various diseases as compared with the susceptibility of the general population of the same ages. It is the more or less unanimous opinion that tuberculosis is the most prevalent cause of death among the feebleminded as a group. We are still unaware of its significance within the specific mental status groups. In order to show a significant relationship between any specific disease and mental defect it is necessary to show that the disease exists in greater proportion among the feebleminded than in the general population of the same age groups. We cannot say that we know this to be true.

In his analysis of 1,000 consecutive deaths at the Earlswood Asylum, Tredgold[2] found that tuberculosis was the cause of death in 39.6 per cent. of the total deaths. Next in order came epileptic convulsions and pneumonia. He states that "excluding tubercle, the nervous system is the part most frequently involved by fatal disease, being followed in order by the respiratory, circulatory, alimentary and urinary systems*."

Shuttleworth and Potts[5] likewise found that mental defectives as a class were unusually prone to the infection of tubercle†. Barr[4], in studying the causes of death in his 625 cases found that tuberculosis came first, with epilepsy, meningitis, diseases of the nervous system, of the heart, of the kidneys, and pneumonia following in order. Again, the U. S. Census Report on feebleminded and epileptics found that the most prominent causes of death were "tuberculosis of lungs, epilepsy, bronchopneumonia, lobar pneumonia, diarrhea and enteritis, influenza, and nephritis. The general death rate per 1,000 under treatment from tuberculosis of the lungs was 5.1 and from epilepsy 4.7. These two diseases cause about one-third of the total number of deaths‡."

From the foregoing it is evident that practically all of our information on causes of death in mental defectives has come from a few scattered articles in the United States and from the earlier work in England of twenty or thirty years ago. Intensive study of American bibliography shows a surprising dearth of articles on mortality among the feebleminded. The only opinions ventured have been simply repe-

*P. 66.
†P. 169.
‡P. 141.
§P. 132.
‖P. 65.

*P. 171.
†P. 199.
‡P. 66.

titions of the material used and quoted by Tredgold.

There is one exception, and that is the 1923 Report of the U. S. Census Bureau. This very excellent report summarizes a total of 1,347 deaths in State schools of the United States for a single year, that of 1922. However, no part of the material can be said to present true mortality rates. The reported death rates have been based upon individuals "under treatment" for a specific year. We have had no rates comparable to the mortality rates for the general population, which are based upon the number of persons exposed to the chance of death for a full year. In the only available figures for the feebleminded we find that patients are counted who have been within the institution a comparatively short time. The cases "under treatment" were determined by adding to the cases actually within the institutions at the end of the year, the number of cases discharged and the number dying during the year. The number dying during the year is then compared with the number "under treatment" and a death rate is calculated. This method produces a larger number of individuals exposed to death for a short time rather than the number of persons exposed for a full year. Likewise, patients discharged within short periods after admission are allowed to weight the group as though present an entire year. Again, according to this method, patients admitted a short time before the end of the year are considered as having been present an entire year. When death rates are determined by this method, it is found that the resulting rate is lower than should be the case, owing to the consideration of the larger number of *persons exposed* to the chance of death for a short time rather than the actual *person-years* involved. It is understood, of course, that the person-year method itself may produce rates which are unjustifiably high where there is a large turnover. In these situations it is proper that the true biologic death rate is somewhat below a person-year death rate although above that obtained on the basis of persons exposed.

Thus, to recapitulate, our knowledge with regard to death rates by age groups, death rates in the various mental status groups and causes of death among the feebleminded is as follows: (1) It has been assumed that the death rate among the feebleminded is two or three times that of the general population and that this rate is highest in the lower grades of mental defect. Previous studies have not taken into account the fact that the population of feebleminded schools is comprised chiefly of the younger age groups and that accurate death rates must be based upon individuals of the same age groups in the general population. (2) The death rate among the feebleminded varies in accordance with the degree of mental defect. (3) The most common causes of death are tuberculosis and epilepsy, the former being particularly high as a cause. We do not yet know whether these causes are more prevalent in any particular mental status group or age group.

PURPOSES

(1) To investigate mortality in mental deficiency for the differing mental status groups and for the various age groups, by sex.

(2) To present a comparison between mortality in mental deficiency and the general population of our State for the same age groups.

(3) To study specific causes of death and determine their numerical and relative importance.

(4) To compare death rates for specific diseases with death rates for the same diseases and among the same age groups in the general population.

MATERIAL

The material of this analysis was based upon a study of 8,976 cases of mental deficiency. These cases were admitted to, were in residence within, or were discharged or died while in residence or on the books of one of our three State schools* between January 1, 1917 and December 31, 1930, inclusive. The information contained herein was abstracted from statistical cards coded directly from individual case records within the three State schools concerned. The data thus obtained were checked for further accuracy from central records in the Department of Mental Diseases. The present paper is in the nature of a preliminary report only. In its entirety the larger study embraces many different phases of mental deficiency, although this section confines itself to a discussion of mortality.

It must be remembered that mentally deficient individuals in State schools are not necessarily typical of mentally deficient individuals in and out of institutions. Mentally defective persons outside of institutions may represent any of the following: (1) They may have been discharged from a State school because their progress has made them self-supporting and able to take their place in society; (2) they may have been discharged because their condition was such that they could be taken care of at home; (3) they may be numbered among the countless other cases of mental deficiency in the community whose mental condition is such that it has never been necessary to send them to a State school. It is possible, also, that the

*The coding of this material from the individual case records was done by a group of eight specially trained assistants who remained in residence at the school concerned until the work was completed. Five and one-half months of work by this group were required to complete the statistical cards used in this study. The data on mortality, of course, constitute only a small portion of the total material available.

The writer wishes to express his appreciation for the coöperation and assistance rendered by the late Dr. George L. Wallace, Superintendent of the Wrentham State School; by Dr. George E. McPherson, Superintendent of the Belchertown State School; and by Dr. Ransom A. Greene, Superintendent of the Walter E. Fernald State School.

numbers discharged each year may be made up of the more favorable cases from the physical aspect, and that mortality in this group may be less than in the groups remaining within State schools.

Again there is the factor of selection in the choice of children who are admitted to our State schools. It is probable that the child who presents a poor physical condition has a better chance for admission. Thus, it is evident that whatever conditions may be found in our sample of State school individuals, they cannot be applied to mentally deficient persons in the general population without considerable reservation.

We may say, however, that our rates are more representative of mental deficiency in the population than has ever been attempted before. This is true because we have included all deaths which have occurred while the persons were under supervision, whether actually within or outside of the school. For example, let us quote a single case. The patient has been admitted to one of our State schools and remains for a period of five years. He then leaves the institution "on parole" and dies a year and a half after leaving the school but before being formally discharged. This is considered as a death within our group. The same is true of patients who die while out of the institution under the administrative classification of "on escape", or "on visit". That is, we have extended our calculations to cover the entire period during which we have had supervision of these persons from the date of admission to the date of final discharge. We have dealt with a total of 878 deaths. Of this total, 110 or 12 per cent. died while out of institutions, a number which is not inconsequential when viewed in relation to the total number of deaths. This method, to a certain extent, removes some of the objections to work which is based upon the observation of only those mentally deficient children who die actually within institutions.

This work was started with a primary population on the books of all State schools as of January 1, 1917. A study was made of all changes in this primary population occurring through new admissions, discharges and deaths up to and including December 31, 1930. The analysis enabled us to determine the changes which had taken place through the addition of new cases by admissions, and the subtraction of cases by means of discharge or death.

METHOD

The quotations as outlined in the introduction indicate that mortality among mental defectives is decidedly higher than in the general population. It was thought that it might be of interest to inquire into the mortality in mental deficiency as a whole and also with reference to the mental status groups idiot, imbecile and moron*. Our first step was to find the specific death rates by age for each of these groups and for both sexes. To do this it was necessary to consider the age at admission, the age at discharge, the age at death, sex and diagnosis. We had reason to feel that these items were fairly accurate in our records.

The specific death rates by age were determined by the "person-year" method† of constructing life tables as set forth in Henderson's Laws of Mortality[6]. The number of cases discharged from State schools within the first five years after admission is large. Although this factor may lead to a somewhat less accurate determination of the population exposed than that determined by any life assurance association where the number of withdrawals in the same year of entrance is small compared with the number of entrants, we feel, nevertheless, that the error introduced is not large.

Throughout the study the sexes are considered

TABLE I

MORTALITY IN IDIOTS COMPARED WITH THE MASSACHUSETTS POPULATION, BY AGE GROUP AND SEX

Age	(1) Aggregate Person Years	(2) Deaths	(3) (2)÷(1) Rate (q_x)	(4) Population Rate Mass. 1910	(5) (3)÷(4) Ratio to Mass. Death Rate
			Males		
2-9	822	53	.0644	.0057	11.2
10-19	2244	59	.0262	.0031	8.4
20-29	1184	62	.0523	.0057	9.1
30-39	542	15	.0276	.0080	3.4
40-49	305	16	.0524	.0121	4.3
50-59	61	5	.0819	.0220	3.7
2-59	5158	210	.0407	.0086	4.7
			Females		
2-9	619	51	.0823	.0051	16.1
10-19	1480	48	.0324	.0027	12.0
20-29	1117	20	.0179	.0048	3.7
30-39	562	12	.0213	.0065	3.2
40-49	176	9	.0511	.0099	5.1
50-59	108	4	.0370	.0176	2.1
2-59	4062	144	.0354	.0074	4.7

*Inclusion in these groups has been determined through the admission intelligence quotient. Uniformity was preserved through the use of the Stanford Revision of the Binet-Simon Tests in all three State schools. The groupings are as follows: Idiot, 0-.24; Imbecile, .25-.49; Moron, .50-.69; Not Mentally Defective, I. Q. .70 plus.

†Modified Formula (pp. 61-62):

$$E_x = \Sigma_0^x n_x - \Sigma_0^{x-1}\theta_x - \Sigma_0^x w_x - \frac{n_x}{2} + \frac{w_x}{2}$$

where n_x denotes the number of new admissions at age x last birthday
where w_x denotes the number of discharges at age x last birthday
where θ_x denotes the number of deaths at age x last birthday
$q_x = \theta_x / E_x$

separately because of the well-known fact that the expectation of life for females is higher than that for males.

MORTALITY IN IDIOTS

Table I presents the average specific mortality rates (q_x) by ten-year age-groupings and for both sexes in cases diagnosed as idiots. The ratios of the death rates to the death rates in the general population of Massachusetts, 1910, Glover's Tables, are also shown. As may be seen, the death rates are highest in the early age groups and gradually approach the rates of the life tables as age advances. It must not be forgotten that the death rates for the early age groups are based upon a small number of observations and, therefore, the possibility of chance variation is large. The same applies to the rates at the end of the life span. However, in the age group 10-19 years, where the rates become fairly stable, we find the death rate among males to be eight times, and among females twelve times, as high as that of the normal population.

For the purpose of comparison it is probably more satisfactory to use the ratios between death rates in our group and death rates in the general population for the same ages. In the males we observe that the ratio for the ages of 2-9 years is eleven times that of the general population. For the ages 10-19 it drops to a ratio of 8. Between 20 and 29 there is a slight rise to a ratio of 9. A sharp drop then occurs to a ratio of 3, which is apparently the same for the age groups 30-39, 40-49 and 50-59. For the entire group between the ages of 2 and 59 years we see that the death rate is approximately five times that of the general population in the same age groups.

For the females we see a little different proportion. The ratios are decidedly higher than the males between the ages of 2 and 20 years, but drop sharply after the age of 20 to a ratio of approximately three times that of the general population. In the age group 2-9 we see a ratio of 16 as opposed to 11 in the males. In the age group 10-19 a ratio of 12 occurs, which is decidedly above the ratio of 8 observed in the males. In the age group 20-29 we see a ratio of 3, which is decidedly lower than the ratio of 9 observed in the males. From the ages of 20 to 50 we note a ratio of approximately 3 for both sexes.

As a general conclusion we may say that, considering the total life span, there is surprisingly little difference between the males and females. Both groups have death rates which are approximately five times that of the general population. However, the death rate in the females is about fourteen times that of the general population between the ages of 2 and 19. It then shows a very marked drop to a ratio of 3 for the remaining years of the life span. The males show a ratio about nine times that of the general population between the ages of 2 and 29, and then show a sharp drop to a ratio of 3 for the remaining age groups.

It appears, then, that female idiots die very rapidly in the ages under 20, but reach a stable rate thereafter, this rate being preserved quite uniformly throughout the older age groups. The males, on the other hand, continue to show high relative death rates up to the age of 30 and reach a stable rate after that age. In other words, idiocy is associated with extremely high death rates for females under 20 years of age. The males have slightly lower rates, but these tend to remain high up to the age of 30 years.

It may be well to mention that in the idiot group we have a large proportion of cases with definite physical abnormalities in addition to the observed mental condition. It is difficult to say what the exact degree of association is between the higher mortality and idiocy. It is reasonable to expect, for example, that a study of hydrocephalus and the various spastic conditions *without mental defect* would also show high death rates. In this situation we cannot be sure that our observed conditions are dependent upon or in any way associated with the lower ranges of mental status. We may be reporting high death rates in children of marked physical defectiveness who happened to have a mental defect in addition.

TABLE II

MORTALITY IN IMBECILES COMPARED WITH THE MASSACHUSETTS POPULATION, BY AGE GROUP AND SEX

Age	(1) Aggregate Person Years	(2) Deaths	(3) (2)÷(1) Rate (q_x)	(4) Population Rate Mass. 1910	(5) (3)÷(4) Ratio to Mass. Death Rate
			Males		
2-9	660	33	.0500	.0057	8.7
10-19	3838	52	.0135	.0031	4.3
20-29	2188	37	.0169	.0057	2.9
30-39	845	31	.0366	.0080	4.5
40-49	422	15	.0355	.0121	2.9
50-59	136	4	.0294	.0220	1.3
2-59	8089	172	.0212	.0086	2.4
			Females		
2-9	482	20	.0414	.0051	8.1
10-19	2814	38	.0135	.0027	5.0
20-29	2451	27	.0110	.0048	2.2
30-39	1481	17	.0114	.0065	1.7
40-49	708	7	.0098	.0099	.9
50-59	362	10	.0276	.0176	1.5
2-59	8298	119	.0143	.0074	1.9

MORTALITY IN IMBECILES

Table II presents the average specific mortality rates (q_x) by ten-year age groupings for

both sexes in cases diagnosed as imbeciles. Ratios of these death rates to the death rates in the general population are also shown. The same general characteristics are observed as was evident among the idiots. That is, the highest rates are observed in the early age groups and gradually approach the rates of the life tables as age advances.

We preserved our method of comparison between the death rates in our group and death rates in the general population for the same ages. In the males the ratio for the age group 2-9 years is eight times that of the general population. In the group 10-19 years it drops to a ratio of 4. Between the ages of 20 and 29 it drops still further to a ratio of 2.9. There is a slight rise in the age group 30-39 to 4.5 and a subsequent drop to a ratio of 2.9 in the age group 40-49. The age group 50-59 reveals a 1.3 ratio. For the entire group between the ages of 2 and 59 years we see that the death rate is 2.4 times that of the general population for the same ages.

Among the females we see essentially the same proportions. In the age group 2-9 we observe a ratio of eight times that of the population. In the group 10-19 years this drops to a ratio of 5. After the age of 20 we see ratios of 2 or less up to the age of 59. The total for this group, or ages between 2 and 59 years, presents a ratio of approximately 2.

There is surprisingly little difference in both the age groups and in the totals for males and females diagnosed as imbeciles. The same general characteristics are evident as were present among the idiots. That is, the highest ratios are observed in the younger age groups, the two groups 2-9 years and 10-19 years. The ratios then tend to stabilize themselves for the age groups 20-59, presenting death rates which are approximately twice that of the general population. In the imbeciles, as in the idiots, the death rates are rather high up to the age of 20, but present little variation for the remaining decades of the life span.

In discussing the sex differences in imbeciles we see very little change in the ratios for the different age groups. The males show a ratio of 8.7 in the group between 2 and 9 years, while the females show a ratio of 8.1. The ratios then decrease rapidly, becoming stable at a ratio of approximately 2 for the age groups 20-29 and higher in both sexes. The ratio of 2.4 for the total males is slightly above that of 1.9 for the total females in the age groups 2-59 years.

We note that the ratios for imbeciles are decidedly lower than those of the idiots. That is, the imbeciles have death rates that are nearer the rates for the general population than the idiots.

MORTALITY IN MORONS

Table III presents the average specific mortality rates (q_x) in ten-year age groupings for both sexes in cases diagnosed as morons. Ratios of the death rates in this group to the death rates in the general population are also shown. The numbers involved in the moron group are larger than either of the two previously presented and, therefore, we can expect that our rates will be more stable. We see here a decidedly different situation in that the observed death rates are very much closer to the population rates and there seems to be very little difference between the ratios for the various age groups. The ratio of the death rate among male morons to the death rate in the general population is 1.8 for the age group 2-9 years. This ratio is 1.2 for the age group 10-19, 1.8 for the age group 20-29, and 1.2 for the age group 30-39. This is in marked contrast to the ratio of population death rates observed in idiots and imbeciles, which were very high in the younger age groups. These ratios rather suggest that whatever the relationship may be between low mental status and high death rate, it is not so operative among the morons, and at least shows no preference for the younger age groups. All age groups appear to be affected approximately the same. The moron under 10 years of age, for example, presents no higher ratio than the moron of 25 years. The ratio for the total age group 2-59 years is .7. That is, all age groups considered, the death rate among morons is lower than that of males in the general population. It is

TABLE III

MORTALITY IN MORONS COMPARED WITH MASSACHUSETTS POPULATION, BY AGE GROUP AND SEX

Age	(1) Aggregate Person Years	(2) Deaths	(3) (2)÷(1) Rate (q_x)	(4) Population Rate Mass. 1910	(5) (3)÷(4) Ratio to Mass. Death Rate
			Males		
2-9	480	5	.0104	.0057	1.8
10-19	5466	22	.0040	.0031	1.2
20-29	1722	18	.0104	.0057	1.8
30-39	396	4	.0101	.0080	1.2
40-49	136	5	.0367	.0121	3.0
50-59	56	0	0	.0220	0
2-59	8256	54	.0065	.0086	.7
			Females		
2-9	227	3	.0132	.0051	2.5
10-19	3032	16	.0052	.0027	1.9
20-29	3625	24	.0066	.0049	1.3
30-39	1407	21	.0149	.0065	2.2
40-49	392	6	.0153	.0099	1.5
50-59	84	6	.0714	.0176	4.0
2-59	8767	76	.0086	.0074	1.1

rather felt that this lower proportion is due to the loading of the higher age groups in the population and the lack of cases among morons in these same high age groups. In all probability it is better to consider the ratios for the individual age groups rather than to base any decision upon the figure for the total.

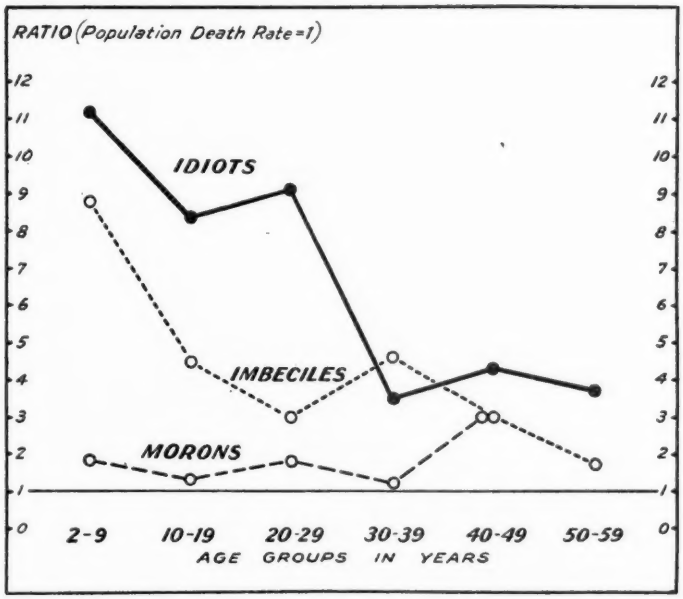
Among the females we see essentially the same characteristics. The ratio for the age group 2-9 years is 2.5, for 10-19 years the ratio is 1.9, and for the age group 20-29 the ratio is 1.3. It shows a slight rise to a ratio of 2.2 in the age group 30-39. The total for all age groups combined is 1.1. Again it is felt that this ratio also is low owing to the fact that the population death rate for the age group 2-59 is loaded in the higher age groups and that the female morons present very few cases in these ages. Therefore, among the females as well as the males it is best to consider the ratios for the individual age groups rather than the ratio for the totals.

In the moron group the death rates are higher than the population rates for the same age groups. However, the ratios are practically constant throughout the various age groups. Whatever the factor may be which results in a higher death rate among morons as compared with the population, it does not appear to exert its effect in any particular age group for either the males or the females. We saw that the idiots and imbeciles were removed from the scene at comparatively young ages, and that very few survived to the older age groups. Morons die at a rate which is higher than the population, but the ratios are practically the same for all age groups. This in itself constitutes an important difference. It would appear, then, that the higher grade cases have a survival rate which is only slightly under that of the population. Idiots and imbeciles are removed from society very rapidly during the early ages. Morons are not removed at the early ages but reach adult life and have chances of surviving which are practically identical for all age groups during the life span.

SEX DIFFERENCES IN MORTALITY FOR THE GROUPS IDIOT, IMBECILE AND MORON

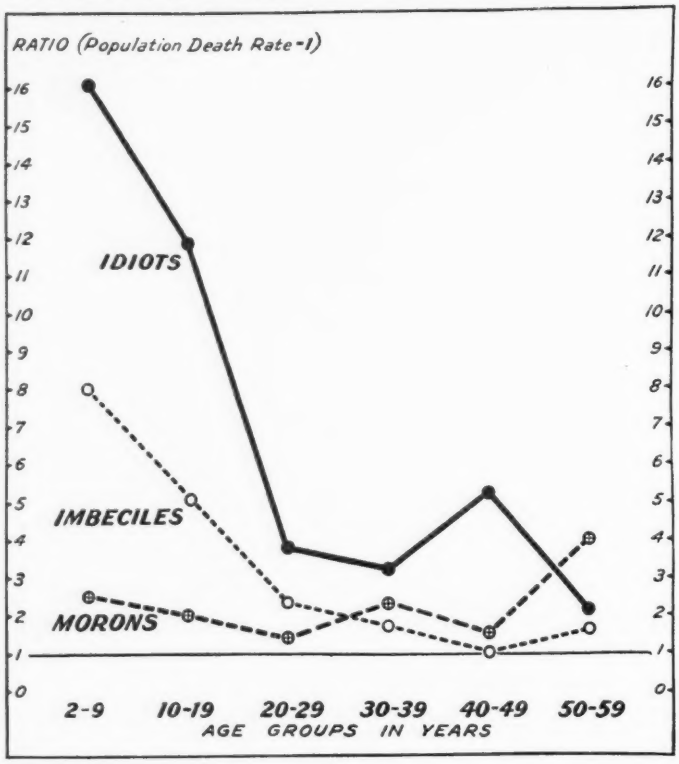
The ratios between mortality in male idiots, imbeciles and morons and in males of the Massachusetts population for the various age groups are outlined in Graph A. This manner of presentation demonstrates the strikingly high ratios in the first decade of life and the manner in which these ratios gradually decrease until they tend to approach each other in the older ages. The male idiots show extremely high ratios for the first three decades or up to the age of 30 years. They then show a rapid drop and for the remaining years of the life span the ratios for these periods do not show a radical departure from the other mental status groups. The male imbeciles also show a high ratio for the first two decades, which falls to a ratio of 3 for the age group 20-29 years. We note that the male morons present ratios which are barely above 1; that is to say, the death rates are not

RATIOS BETWEEN MORTALITY IN MALE IDIOTS, IMBECILES AND MORONS IN MASSACHUSETTS STATE SCHOOLS, 1917-1930, AND MORTALITY IN MALES OF MASS. POPULATION, BY AGE GROUPS.



GRAPH A.

RATIOS BETWEEN MORTALITY IN FEMALE IDIOTS, IMBECILES AND MORONS IN MASS. STATE SCHOOLS, 1917-1930, AND MORTALITY IN FEMALES OF MASS. POPULATION. — — BY AGE GROUPS.



GRAPH B.

greatly above those of the general population.

Graph B presents the ratios between mortality in female idiots, imbeciles and morons and mortality in females of the Massachusetts population. Among the female idiots the ratios of 16 and 12 for the first two decades of life are extremely high. For the age group 20-29 there is a marked drop to a ratio of under 4, and this is preserved for the remaining age groups. The imbeciles also present high ratios for the first two decades, dropping to a ratio of 2 for the age group 20-29 years. The morons have rather low ratios for all age groups, and, as a matter of fact, are not greatly above the death rates of the general population, the ratio being about two times that of the population death rates. We note in Graph B that the high ratios for female idiots and imbeciles are observed before the age of 20 years. From that age onward the ratios are comparatively low. This fact is to be noted as it is rather in contrast with the condition noted among the males, who show rather high ratios up to the age of 30 years.

MORTALITY IN MENTAL DEFECTIVES

Table IV gives the average specific mortality rates (q_x) by ten-year age groupings for both sexes in the total of all cases diagnosed as idiots, imbeciles or morons. Ratios of the death rates in this group to the death rates in the general population are presented in Table IV and Graph C. In so far as this table is based on the

TABLE IV

MORTALITY IN MENTAL DEFECTIVES (FEEBLEMINDED) COMPARED WITH THE MASSACHUSETTS POPULATION, BY AGE GROUP AND SEX

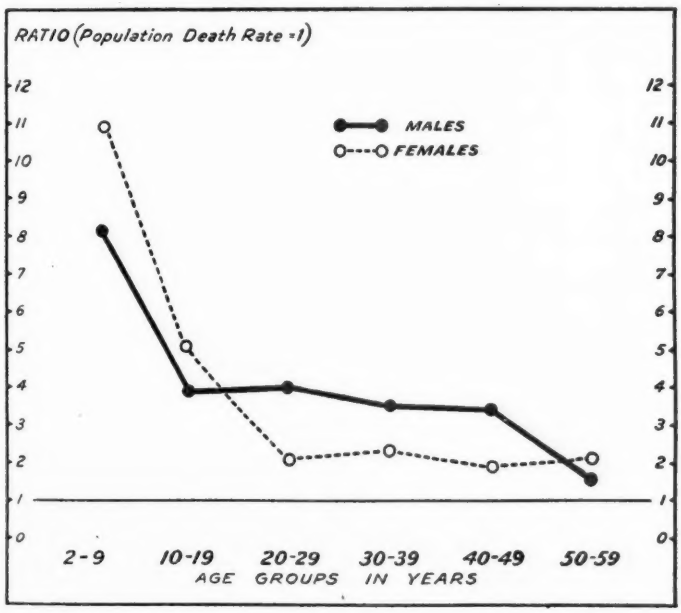
Age	(1) Aggregate Person Years	(2) Deaths	(3) (2)÷(1) Rate (q_x)	(4) Population Rate Mass. 1910	(5) (3)÷(4) Ratio to Mass. Death Rate
			Males		
2-9	1968	91	.0462	.0057	8.1
10-19	11548	133	.0115	.0031	3.7
20-29	5090	117	.0229	.0057	4.0
30-39	1783	50	.0280	.0080	3.5
40-49	863	36	.0417	.0121	3.4
50-59	253	9	.0356	.0220	1.6
2-59	21505	436	.0202	.0086	2.3
			Females		
2-9	1328	74	.0557	.0051	10.9
10-19	7326	102	.0139	.0027	5.1
20-29	7193	71	.0098	.0048	2.0
30-39	3450	50	.0145	.0065	2.2
40-49	1276	22	.0172	.0099	1.7
50-59	554	20	.0361	.0176	2.0
2-59	21127	339	.0160	.0074	2.1

totals of all three preceding groups idiots, imbeciles and morons, the numbers are very much larger and, consequently, the findings will present fewer chance variations. In interpreting these results for *all mental defectives* it should be recalled that in all probability our institution sample comprises larger proportions of idiots and imbeciles than would be true in a population sample of mental defectives. Consequently, the death rates which we observe in Table IV for the entire group are probably higher than would be found in a population sample of mental defect. This is explained by the relatively lower death rates among the morons and the extremely high death rates among the imbeciles and particularly the idiots. The population sample would probably contain a larger proportion of morons, and for that reason the rates for the population sample would be lower than those of our institution samples.

The ratio for the total of all age groups is 2.3 for the males and 2.1 for the females. There is very little difference in these totals. However, one important point must be mentioned again lest anyone make the error of placing too great reliance in these totals for all age groups. As was brought out before, deaths in the population increase markedly as the older age groups are approached, while the deaths in our group tend to taper off as the older age groups are approached. This means that mortality for the age group 2-59 years in the general population is loaded in the higher age groups so that the rate for this total is higher than it should be. That is, it is higher than it should be for a fair comparison with the total rate for mental defectives dying in the age group 2-59 years. Consequently, it is much better to place more dependence on the rates for the individual age groups than for the totals for all age groups.

The sex differences are outlined in Graph C. We observe that the ratio for the males is 8.1 for the age group 2-9, and then drops to 3.7 for the age group 10-19. The latter ratio is then preserved quite uniformly for the remaining age groups studied. Among the females, however, we observe that the ratio for the age group 2-9 years was approximately eleven times that of the population. This drops to 5.1 in the age group 10-19 and then to a figure of 2 for the age group 20-29. This ratio of 2 is preserved for the remaining age groups studied. Thus, we observe that the females have rather high relative death rates up to the age of 20 and a much lower death rate for the remaining years of the life span. The males have a rather high death rate under 10 years, although it is a third lower than that of the females, and then reaches a rather stable ratio for the remaining age groups. This rather suggests to us that the physical abnormalities, actual or potential, existing in female mental defectives are more likely to cause early death than among males. That is, the possibility of early death is greater among the females than among the males. The males have a fairly high death rate for ages under 10, but reach a very stable rate for ages over 10. The female is subjected to a much

RATIOS BETWEEN MORTALITY IN TOTAL MENTAL DEFECTIVES IN MASS. STATE SCHOOLS, 1917-1930, AND MORTALITY IN MASS. POPULATION, BY AGE GROUPS AND SEX.



GRAPH C.

greater chance of death in the younger age groups under 20 than the male, but if she survives to the age of 20 she has a greater chance of living through the higher ages. The males, on the other hand, are not subjected to as great a chance of death under 10 years, but must face a fairly high death rate throughout the remaining years of the life span.

In summarizing, we may say that the tendency is for female mental defectives to die under the age of 20, but those who survive have a fairly good chance of living to older ages. The ratio for these higher age groups is approximately two times that of the population. The ratio for the entire group 2-59 years is 2.1. The males, on the other hand, do not die as rapidly under the age of 10 but have a fairly high ratio of 3.5 throughout the remaining years of the life span. The ratio for the group as a whole, 2-59 years, is 2.3.

COMPARISON BETWEEN DEATH RATES IN MENTAL DEFICIENCY FOR MASSACHUSETTS FOR THE PERIOD 1917-1930 COMPARED WITH DEATH RATES IN MENTAL DEFICIENCY FOR THE ENTIRE COUNTRY—1922

In the preceding sections we have outlined the comparison between death rates in mental defect and the general population of our State. It is quite obvious that death rates in mental defect are decidedly above the rates of the general population for Massachusetts. However, we may ask at this point how these rates compare with death rates for other sections of the country. We have certain material which is not exactly comparable but has the distinction of being the only data available. This is found in the U. S. Census Report on Feebleminded for 1923. As has been mentioned before in this paper, the rates calculated in this publication are based upon cases "under treatment". In this manner a larger number of cases are considered as having been exposed to chance of death than is actually the case. With a fictitiously large number of cases "under treatment" the resulting death rate will be lower than is actually the case. In other words, if a more exact method had been used, the resulting mortality rates would have been higher than the rates which have been presented to us. This fact should be borne in mind before we attempt a comparison with the mortality rates evolved in this study. Again it should be recalled that the Census study is based upon a total of 1,347 deaths throughout the United States for the year 1922, and that the Massachusetts study is based upon a total of 878 deaths over a fourteen-year period, 1917-1930, within a single State. The Census study has the advantage of being a country-wide sample and presents results for the country as a whole. It has the disadvantage of recording death rates from State units which have widely different standards of administration and medical care. For example, we could expect widely differing death rates between States caring only for the most urgent cases of mental defect as compared with States carrying out a comprehensive training program for all mental defectives coming to their attention; or between States providing a minimum number of physicians as compared with States providing thorough and complete medical care. The Massachusetts study has the advantage of extending over a period of fourteen years as contrasted to a single year of the Census study, and presents results which were obtained through the operation of a system exercising rigid administrative control and maintaining a very definite standard of medical care. The Massachusetts study has the disadvantage of being slightly smaller in numbers, 878 cases as compared with 1,347 in the Census study, and it represents the results of but a single State unit.

TABLE V

PATIENTS WHO DIED IN INSTITUTIONS FOR MENTAL DEFECTIVES (FEEBLEMINDED) THROUGHOUT THE UNITED STATES DURING 1922, RATE PER 1,000 UNDER TREATMENT, COMPARED WITH MORTALITY IN MASSACHUSETTS STATE SCHOOLS FOR THE PERIOD 1917-1930, BY MENTAL STATUS GROUP AND SEX

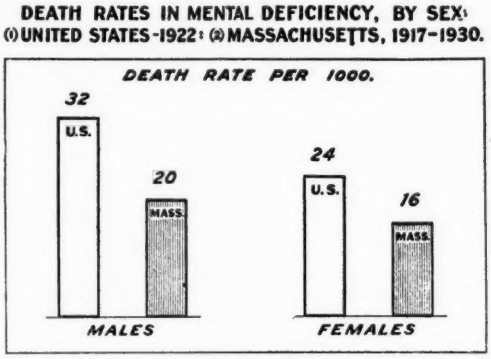
Mental Status Group	Source	Death Rate per 1,000 Male	Female
Total—All Groups	United States (Census Study)	32.2	24.8
	Massachusetts (Present Study)	20.5	16.2
Idiot	United States	80.0	56.8
	Massachusetts	40.7	35.4
Imbecile	United States	29.0	24.2
	Massachusetts	21.2	14.3
Moron	United States	11.6	8.9
	Massachusetts	6.5	8.6
Not Mentally Defective (Not F. M.)	United States	18.7	29.6
	Massachusetts	6.5	6.1
Unknown	United States	45.9	63.6
	Massachusetts	133.8	294.0
Unclassified	United States	53.9	65.8

Although the different methods used in obtaining the death rates for these two groups invalidate a comparison to a certain extent, at the same time it is felt that the available material is too valuable to be discarded entirely. Table V outlines the data as presented.

We find that the recorded death rates for the country as a whole are definitely higher than those of the Massachusetts study. If we take the total for all groups, we observe that the death rate in males was 32.2 per 1,000 in the Census study and 20.5 in the Massachusetts study. For the females the Census study presented a death rate of 24.8 per 1,000 as com-

pared with 16.2 for the Massachusetts study (Graph D). Although in the Census study a method was used which produced lower rates than are actually the case, the Massachusetts mortality rates are definitely under those of the Census study.

When we turn to the individual mental status groups idiot, imbecile and moron, we see essentially the same condition. In all groups and for both sexes we see that the Massachusetts mortality rates are definitely lower than the Census rates for the entire country. In the group "Not Mentally Defective" (Not Feeble-



GRAPH D.

minded) we see that the ratios are even larger. In this group the Census study presents a death rate of 18.7 for males and 29.6 for females. The Massachusetts study, on the other hand, presents a death rate of 6.5 for males and 6.1 for females. The group in which the mental status was "Unknown" presents the only exception to this general tendency. Here we observe that the death rates in the Massachusetts study are higher than in the Census study. It is rather difficult to explain this difference. A possible explanation which presents itself at this time is that the "Unknowns" in the Census study may comprise cases of higher mental grade. It is a well-known fact that mental testing has not become an accepted procedure in some States and that the diagnoses are made upon less definite criteria. The Massachusetts "Unknowns," however, would be made up of a group in which it had been impossible to obtain a mental rating. Quite obviously, this group would be made up of the lowest grade cases in the school. Where less complete machinery is available for the testing of children it is possible to conceive that higher grade cases may have been included in the "Unknown" group in the collection of data from the entire country. In so far as the numbers involved in these groups are small, it probably is not worth while to go deeper into the matter.

Thus, the Massachusetts mortality rates are definitely below the death rates for the entire country. In previous sections we demonstrated that death rates in mental defect were extremely high as compared with the general population in our State. These extremely high death rates were somewhat disturbing and gave rise to considerable conjecture as to the causes. In this situation it is probably not untimely to show that a similar situation obtains throughout the country. We observe that a comparison with the admittedly low rates for the entire country, as outlined in the 1923 Census Report shows that Massachusetts has a death rate in mental deficiency which is nearly 40 per cent. lower than that of the United States as a whole.

PRACTICAL APPLICATION OF THE OBSERVED DEATH RATES

Up to this point we have presented material on actual death rates, and have compared these with figures for the general population. It is rather difficult for one not thoroughly familiar with the various procedures of vital statistics to gain any great amount of information from these tables. It was thought best, therefore, to insert a section outlining the number of survivors which we could expect at the various ages if we applied the observed death rates for each decade, taking an initial population of 1,000 persons born in each group.

In the following sections we outline this material for each mental status group idiot, imbecile, moron and the mentally defective group as a whole. We also present a population comparison. The material is presented in two ways: First, we start with an initial population of 1,000 persons born and then apply the death rates for each age group by decades. That is, we determine the number of persons who would die in the first ten years of life, applying the death rates for the specific group in the first ten years of life. As our numbers were not sufficiently large, it was impracticable to attempt to present a death rate for each individual year. The rates used are for ten-year periods. In each group we have started with 1,000 persons born and have applied the death rates for each ten-year period up to 60 years of age. Thus, we have the number of persons surviving in the original group of 1,000 at the specific ages of 10, 20, 30, 40, 50 and 60 years. This presents the material in a simple manner and shows the exact number of survivors which we may expect to have at the end of each ten-year period, or at the ages specified.

In the tables presenting the material on survivors in each 1,000 persons born it has been necessary to make an estimate of the death rates for the period from birth to two years. In so far as we had no cases within our State schools under two years of age and, therefore, no deaths, we had no way of obtaining rates for this period. It became necessary, then, to estimate a rate for the period 0-9 years based on the observed rate between 2 and 9 years. We did this by

applying approximately the same difference in rates observed between the 0-2 year group and the 2-9 year group in the general population. This method was not entirely satisfactory, however, as we had reason to believe that death rates among certain mentally defective groups might be much higher in the first two years of life and our estimated rates could be considered only as a rough approximation.

To obviate justified criticism of the above method we have also prepared the material by excluding the estimated rates for the first two years of life. In this method we took simply the actual observed rates for the ages 2-9 years. We did this by starting with our original group of 1,000 at the age of two years instead of the time of birth. This is probably a more reliable method than the other. Both sets of tables are herein presented. Similar data for both sexes in the general population are also presented for comparison, using both methods as outlined.

TABLE VI

NUMBERS SURVIVING AT CERTAIN AGES OF EACH 1,000 MALE IDIOTS BORN AND ALSO OF EACH 1,000 AGED 2 YEARS

Age	Numbers Surviving at Certain Ages of Each 1,000 Born			
	Number Alive at Beginning of Period	Annual Death Rate	Deaths During Period	Deaths Accumulated to End of Period
0-9	1000	.1209	724	724
10-19	276	.0262	65	789
20-29	211	.0523	87	876
30-39	124	.0276	31	907
40-49	93	.0524	38	945
50-59	55	.0819	32	977
60	23			
Age	Numbers Surviving at Certain Ages of Each 1,000 Aged 2 Years			
2-9	1000	.0644	413	413
10-19	587	.0262	137	550
20-29	450	.0523	187	737
30-39	263	.0276	64	801
40-49	199	.0524	86	887
50-59	113	.0819	65	952
60	48			

In studying this material for the various mental status groups and for each sex we have arranged the data in the manner outlined in Table VI. This is used as an example, and outlines the material for idiot males only. In applying the observed death rates for all groups above 10 years of age, and using the observed death rate between 2 and 9 years plus an estimated death rate for 0-2 years, we see that out of 1,000 male idiots born we can expect that but 276 will survive to the age of 10 years; 211 would be alive at the age of 20 years; 124 would survive to the age of 30 years; 93 would be living at 40 years; 55 at the age of 50 years; and but 23 would survive to the age of 60 years. Thus, out of 1,000 male idiots born, but 23 would be alive at the age of 60 years.

In considering the other method, that of studying the number of survivors at certain ages for each 1,000 male idiots at the age of 2 years, we get results that are different but not markedly so. Thus, of 1,000 idiots living at the age of 2 years, 587 would survive to the age of 10 years; 450 would be alive at the age of 20; 263 at the age of 30; 199 at 40 years of age; 113 at the age of 50; and 48 at the age of 60 years. It probably is well to repeat at this time that the results of the first section of this table "Numbers Surviving at Certain Ages of Each 1,000 Born" is probably nearer the actual truth of the matter. At the same time the results of section 2 "Numbers Surviving at Certain Ages of Each 1,000 Aged 2 Years" are more accurate, inasmuch as they are based upon actual material and not upon estimation. This method of analysis was applied to each mental status group by sex. However, individual tables are not being presented but simply a summary made. These summaries are based on both methods.

SURVIVORS OF EACH 1,000 MALE MENTAL DEFECTIVES BORN AND OF EACH 1,000 AGED TWO YEARS, AT CERTAIN AGES

In the previous section we outlined the method whereby we obtained the numbers of survivors for various ages in each 1,000 male idiots born, and again in each 1,000 male idiots aged two years. At this point we wish to proceed to apply this method to the other mental status groups within the various ages and also to make a comparison with the number of survivors which we could expect in the Massachusetts population of the same age groups. To make this comparison possible we have started with 1,000 persons born and also 1,000 persons aged two years in the general population of Massachusetts. To these original groups of 1,000 we have applied the death rates which have occurred in this State for the same period of years. Thus, we have carried along for the various age groups a comparison between the number of survivors that we might expect among the idiots, imbeciles, morons, total mental defectives and also in the Massachusetts population.

Table VII and Graph E present this material for the males. In the first section of this table (based on each 1,000 persons born) we observe that the number of survivors among the morons for each ten-year period is closest to the population figures, and that the lower we go in the intelligence, the greater the divergence from the number of survivors in the population. Thus, at the age of ten we observe that out of 1,000 persons born in each group 276 idiots have survived, 314 imbeciles, and 705 morons, while out

of the original 1,000 persons born in the general population 803 are still alive. The decrease between the ages of 10 and 20 is not marked, but at the age of 30 we find that a noticeable reduction has occurred, particularly in the cases of lower mental grade. We observed at the age of 30 that out of the original 1,000 born in each group 124 idiots have survived, 231 imbeciles, 610 morons, and 739 in the general population. At the age of 50 we find that but 55 idiots have survived, 111 imbeciles, 379 morons, and 604 in the general population. At the age of 60 we note that our groups are practically non-existent. Of the original 1,000 born 23 idiots have survived and 82 imbeciles, while in the general population 482 persons have survived. In this table we also present a column showing the expected survivors for all three of these groups together or for "total mental defectives".

The second half of this table presents the number of survivors at certain ages of each 1,000 male mental defectives aged two years. This is the second method, as mentioned previously, and has the advantage of being more exact as it is based upon actual observed deaths. We find that by discarding the first two years of life for our groups and for the general population we obtain figures which are somewhat different from the results based upon each 1,000 cases born. However, the final results do not differ greatly. At the age of 60 but 48 idiots and 174 imbeciles have survived as compared with a figure of 575 persons for the general population.

TABLE VII

NUMBERS SURVIVING AT CERTAIN AGES OF EACH 1,000 MALE MENTAL DEFECTIVES BORN AND OF EACH 1,000 AGED 2 YEARS, BY MENTAL STATUS GROUP, COMPARED WITH MASSACHUSETTS POPULATION

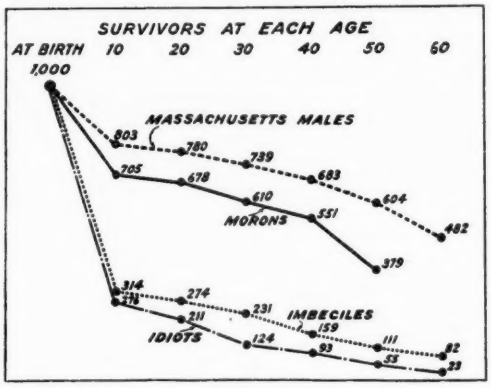
Each 1,000 Males Born

Age	Number Alive at Beginning of Period				
	Total Mental Defectives	Idiots	Imbeciles	Morons	Massachusetts Population
0-9	1000	1000	1000	1000	1000
10-19	325	276	314	705	803
20-29	290	211	274	678	780
30-39	230	124	231	610	739
40-49	173	93	159	551	683
50-59	113	55	111	379	604
60	79	23	82	—	482

Each 1,000 Males Aged 2 Years

Age	Number Alive at Beginning of Period				
	Total Mental Defectives	Idiots	Imbeciles	Morons	Massachusetts Population
2-9	1000	1000	1000	1000	1000
10-19	685	587	663	920	956
20-29	610	450	579	884	930
30-39	484	263	488	797	881
40-49	364	199	336	719	814
50-59	238	113	234	495	720
60-	166	48	174	—	575

NUMBERS SURVIVING AT CERTAIN AGES OF EACH 1,000 MALE IDIOTS, IMBECILES AND MORONS BORN COMPARED WITH MALE SURVIVORS IN MASSACHUSETTS POPULATION.



GRAPH E.

SURVIVORS OF EACH 1,000 FEMALE MENTAL DEFECTIVES BORN AND OF EACH 1,000 AGED TWO YEARS

Table VIII and Graph F present the same material for the females. Here we observe that of each 1,000 persons born 268 idiots, 387 imbeciles and 722 morons survived at the age of 10 years as compared with 831 persons of each 1,000 born in the Massachusetts population. At the age of 30 we find that 161 idiots, 302 imbeciles and 641 morons survived as compared with 772

TABLE VIII

NUMBERS SURVIVING AT CERTAIN AGES OF EACH 1,000 FEMALE MENTAL DEFECTIVES BORN AND OF EACH 1,000 AGED 2 YEARS, BY MENTAL STATUS GROUP, COMPARED WITH MASSACHUSETTS POPULATION

Each 1,000 Females Born

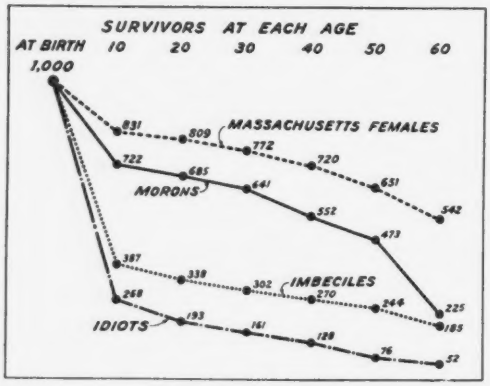
Age	Number Alive at Beginning of Period				
	Total Mental Defectives	Idiots	Imbeciles	Morons	Massachusetts Population
0-9	1000	1000	1000	1000	1000
10-19	341	268	387	722	831
20-29	296	198	338	685	809
30-39	268	161	302	641	772
40-49	232	128	270	552	720
50-59	195	76	244	473	651
60-	135	52	185	225	542

Each 1,000 Females Aged 2 Years

Age	Number Alive at Beginning of Period				
	Total Mental Defectives	Idiots	Imbeciles	Morons	Massachusetts Population
2-9	1000	1000	1000	1000	1000
10-19	632	503	713	899	962
20-29	550	362	622	854	937
30-39	498	302	557	799	894
40-49	430	241	497	687	834
50-59	362	143	450	589	754
60	250	98	325	281	628

persons in the general population. At the age of 50 we note that the survivors have been reduced to 76 idiots, 244 imbeciles and 473 morons, while 651 of the population were still living. At the age of 60 we observe that 52 idiots, 185 imbeciles and 225 morons survived as compared with a figure of 542 for the general population of this age.

NUMBERS SURVIVING AT CERTAIN AGES OF EACH 1000 FEMALE IDIOTS, IMBECILES AND MORONS BORN COMPARED WITH FEMALE SURVIVORS IN MASSACHUSETTS POPULATION.



GRAPH F.

Considering the female group by the second manner of this comparison, that is, by taking 1,000 female mental defectives aged two years and making a comparison with the general population, we see figures which correspond rather closely to the situation developed in the males. As is to be expected, the numbers of survivors are decidedly larger but are greatly over-shadowed by the figures for the general population. At the age of 60 we note that out of the original 1,000 aged 2 years 98 idiots, 325 imbeciles and 281 morons had survived as compared with 628 in the general population.*

SEX DIFFERENCES IN NUMBERS OF SURVIVORS

Interesting differences in the numbers of survivors for the two sexes are observed, and this material is presented in Table IX. Here we study the numbers surviving at certain ages for each 1,000 mental defectives born compared with the Massachusetts population by sex. We observe that in each decade fewer males survive than females. In so far as this is also true in the general population, it becomes necessary to devise some means of comparing our differences with the population differences. We have done this by calculating a ratio of male survivors to female survivors. The resulting ratios are presented in Table IX. We note that not only is the number of survivors quite small among the males as compared with the females for each age group but the ratios of female survivors over male survivors increase decidedly as the older ages are approached. Thus, the ratio of female survivors to male survivors at the age of 10 is 1.04; at the age of 30 this is increased to 1.16; at 40 it is 1.34; at 50 it is increased to 1.72; and at 60 it is 1.70. These figures are quite significant in so far as we see that the sex ratio for survivors in the general population ranges between 1.03 and 1.12. Thus, we may say quite definitely from this material that the male mental defectives have definitely lower chances of survival and that this survival chance is less than it is for males in the general population.

*The higher rate of survival for imbeciles over morons is probably a statistical accident. The death rates for the groups 50-59 years are based upon very small numbers.

TABLE IX

NUMBERS SURVIVING AT CERTAIN AGES FOR EACH 1,000 MENTAL DEFECTIVES BORN COMPARED WITH MASSACHUSETTS POPULATION, BY SEX

Each 1,000 Born

Age	Number Alive at Beginning of Period					
	Mental Defectives			Massachusetts Population		
	Male	Female	Ratio Males =1	Male	Female	Ratio Males =1
0-9	1000	1000	1.0	1000	1000	1.0
10-19	325	341	1.04	803	831	1.03
20-29	290	296	1.02	780	809	1.03
30-39	230	268	1.16	739	772	1.04
40-49	173	232	1.34	683	720	1.05
50-59	113	195	1.72	604	651	1.07
60	79	135	1.70	482	542	1.12

CAUSES OF DEATH IN MENTAL DEFECTIVES

In the foregoing sections we have discussed death rates for the various mental status groups and have compared them with death rates in the general population. At this point we wish to turn to the individual causes of death and to study the particular diseases involved in deaths occurring in our group.

Table X presents this information. Here we have the actual causes of death in the groups idiot, imbecile, moron, not mentally defective, and the group in which the mental status was unknown. A total is presented for all mental defectives; that is, the group comprised of idiots, imbeciles and morons. A second total is presented for all groups together; that is, the mentally defective together with the group not mentally defective and those in whom the mental status was unknown. The numbers in the groups not mentally defective and mental status unknown are rather small and will not be discussed.

The diseases which make up 1 per cent. or more of the total deaths among mental defectives (feebleminded) are presented in the order of their importance in Table XI and Graph G. Thus, the most important causes of death among the

Causes of Death	Total—All Deaths (3) + (4) + (5) + (6) + (7)						Total—Deaths in Mental Defecti (3) + (4) + (			
	Total		Male		Female		Total		Male	
	No.	%	No.	%	No.	%	No.	%	No.	%
Epidemic, Endemic and Infectious Diseases										
Measles	30	3.4	15	3.0	15	3.9	30	3.9	15	3.4
Scarlet Fever	5	.6	2	.4	3	.8	5	.6	2	.5
Whooping Cough	3	.3	2	.4	1	.3	2	.3	2	.5
Diphtheria	11	1.3	6	1.2	5	1.3	8	1.0	4	.9
Influenza	142	16.2	92	18.6	50	13.1	127	16.4	79	18.1
Dysentery	2	.3	1	.2	1	.3	2	.3	1	.2
Erysipelas	1	.1	1	.2	—	—	1	.1	1	.2
Acute Poliomyelitis	1	.1	—	—	1	.3	1	.1	—	—
Lethargic Encephalitis	2	.3	—	—	2	.5	2	.3	—	—
Tuberculosis of Lungs	126	14.3	68	13.8	58	15.2	110	14.2	59	13.5
Other Forms of Tuberculosis	35	4.0	23	4.7	12	3.1	32	4.1	21	4.8
Syphilis (Non-nervous)	5	.6	3	.6	2	.5	5	.6	3	.7
General Diseases										
Cancer	3	.3	1	.2	2	.5	2	.3	1	.2
Tumor (Non-cancerous)	1	.1	—	—	1	.3	1	.1	—	—
Pellagra	5	.6	4	.8	1	.3	4	.5	4	.9
Other General Diseases*	27	3.1	15	3.0	12	3.1	25	3.2	12	2.8
Diseases of Nervous System										
Cerebrospinal meningitis	7	.8	1	.2	6	1.6	5	.6	1	.2
Diseases of Spinal Cord	9	1.0	7	1.4	2	.5	8	1.0	6	1.4
Apoplexy (Cerebral Hemorrhage)	17	1.9	8	1.6	9	2.3	15	1.9	6	1.4
General Paralysis of Insane	9	1.0	4	.8	5	1.3	9	1.2	4	.9
Other Diseases of Brain	1	.1	—	—	1	.3	1	.1	—	—
Epilepsy	39	4.4	20	4.0	19	4.9	33	4.3	16	3.7
Other Diseases of Nervous System	10	1.1	5	1.0	5	1.3	7	.9	4	.9
Diseases of Circulatory System										
Endocarditis and Myocarditis	7	.8	2	.4	5	1.3	7	.9	2	.5
Other Diseases of the Heart	41	4.6	24	4.9	17	4.5	36	4.7	22	5.0
Other Diseases of Circulatory System	5	.6	2	.4	3	.8	3	.4	1	.2
Diseases of Respiratory System										
Bronchitis	9	1.0	5	1.0	4	1.0	7	.9	4	.9
Bronchopneumonia	78	8.9	38	7.7	40	10.5	67	8.7	32	7.3
Lobar Pneumonia	60	6.8	39	7.9	21	5.5	57	7.4	37	8.5
Pleurisy	5	.6	5	1.0	—	—	4	.5	4	.9
Gangrene of Lungs	2	.3	1	.2	1	.3	2	.3	1	.2
Other Diseases of Respiratory System	11	1.3	5	1.0	6	1.6	10	1.3	4	.9
Diseases of Digestive System										
Ulcer of Stomach	5	.6	4	.8	1	.3	4	.5	4	.9
Other Diseases of Stomach (Cancer Excluded)	3	.3	1	.2	2	.5	3	.4	1	.2
Diarrhea and Enteritis	35	4.0	21	4.2	14	3.7	32	4.1	20	4.6
Appendicitis and Typhlitis	5	.6	4	.8	1	.3	5	.6	4	.9
Hernia and Intestinal Obstruction	8	.9	6	1.2	2	.5	7	.9	6	1.4
Other Diseases of Intestines	4	.5	4	.8	—	—	4	.5	4	.9
Other Diseases of Liver	1	.1	—	—	1	.3	1	.1	—	—
Other Diseases of Digestive System	8	.9	6	1.2	2	.5	7	.9	6	1.4
Diseases of Genito-Urinary System										
Acute Nephritis	3	.3	2	.4	1	.3	3	.4	2	.5
Chronic Nephritis	19	2.2	6	1.2	13	3.4	16	2.1	6	1.4
Other Diseases of Kidneys	5	.6	1	.2	4	1.0	4	.5	1	.2
Diseases of Skin										
Gangrene	1	.1	1	.2	—	—	1	.1	1	.2
Other Diseases of Skin	1	.1	1	.2	—	—	1	.1	1	.2
Diseases of Bones and Locomotor System										
Diseases of Bones	1	.1	1	.2	—	—	1	.1	1	.2
Malformations	16	1.8	12	2.4	4	1.0	14	1.8	10	2.3
Violence										
Accidental Traumatism	3	.3	3	.6	—	—	2	.3	2	.5
Other External Violence	12	1.4	10	2.0	2	.5	9	1.2	8	1.8
Cause of Death Unknown†	39	4.4	14	2.8	25	6.5	33	4.3	12	2.8
TOTAL	878	100.0	496	100.0	382	100.0	775	100.0	437	100.0

TABLE X

ES OCCURRING IN MASSACHUSETTS STATE SCHOOLS, 1917-1930, BY MENTAL STATUS AND SEX: NUMBERS AND PERCENTAGE DISTRIBUTIO

3 4 5

(Feebleminded)		Idiots 0-.24 I. Q.						Imbeciles .25-.49 I. Q.						Morons .50-.69 I. Q.			
Female		Total		Male		Female		Total		Male		Female		Total		Male	
No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
15	4.4	19	5.4	10	4.7	9	6.2	8	2.8	4	2.3	4	3.4	3	2.3	1	1.9
3	.9	4	1.1	2	.9	2	1.4	1	.3	—	—	1	.9	—	—	—	—
—	—	1	.3	1	.5	—	—	—	—	—	—	—	—	1	.8	1	1.9
4	1.2	6	1.7	2	.9	4	2.8	2	.7	2	1.2	—	—	—	—	—	—
48	14.2	57	16.1	36	17.1	21	14.6	45	15.6	30	17.4	15	12.6	25	19.2	13	24.0
1	.3	2	.6	1	.5	1	.7	—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	1	.3	1	.6	—	—	—	—	—	—
1	.3	—	—	—	—	—	—	—	—	—	—	—	—	1	.8	—	—
2	.6	—	—	—	—	—	—	1	.3	—	—	1	.9	1	.8	—	—
51	15.1	53	14.9	37	17.6	16	11.1	37	12.9	17	9.9	20	16.8	20	15.4	5	9.2
11	3.3	15	4.2	12	5.7	3	2.0	11	3.9	6	3.5	5	4.2	6	4.6	3	5.5
2	.6	3	.8	2	.9	1	.7	1	.3	—	—	1	.9	1	.8	1	1.9
1	.3	1	.3	1	.5	—	—	1	.3	—	—	1	.9	—	—	—	—
1	.3	1	.3	—	—	1	.7	—	—	—	—	—	—	—	—	—	—
—	—	4	1.1	4	1.9	—	—	—	—	—	—	—	—	—	—	—	—
13	3.8	12	3.4	4	1.9	8	5.6	7	2.4	3	1.7	4	3.4	6	4.6	5	9.2
4	1.2	3	.8	1	.5	2	1.4	1	.3	—	—	1	.9	1	.8	—	—
2	.6	3	.8	1	.5	2	1.4	3	1.0	3	1.7	—	—	2	1.5	2	3.7
9	2.7	4	1.1	3	1.4	1	.7	7	2.4	2	1.2	5	4.2	4	3.1	1	1.9
5	1.5	2	.6	2	.9	—	—	5	1.7	—	—	5	4.2	2	1.5	2	3.7
1	.3	1	.3	—	—	1	.7	—	—	—	—	—	—	—	—	—	—
17	5.0	25	7.0	11	5.2	14	9.7	5	1.7	4	2.3	1	.9	3	2.3	1	1.9
3	.9	4	1.1	3	1.4	1	.7	1	.3	—	—	1	.9	2	1.5	1	1.9
5	1.5	1	.3	—	—	1	.7	5	1.7	2	1.2	3	2.5	1	.8	—	—
14	4.1	9	2.5	6	2.8	3	2.0	20	7.0	13	7.5	7	5.9	7	5.4	3	5.5
2	.6	—	—	—	—	—	—	3	1.0	1	.6	2	1.7	—	—	—	—
3	.9	2	.6	1	.5	1	.7	5	1.7	3	1.7	2	1.7	—	—	—	—
35	10.3	44	12.4	19	9.0	25	17.4	20	7.0	13	7.5	7	5.9	3	2.3	—	—
20	5.9	23	6.5	16	7.6	7	4.9	28	9.7	17	9.9	11	9.2	6	4.6	4	7.3
—	—	2	.6	2	.9	—	—	2	.7	2	1.2	—	—	—	—	—	—
1	.3	1	.3	1	.5	—	—	1	.3	—	—	1	.9	—	—	—	—
6	1.8	7	2.0	3	1.4	4	2.8	3	1.0	1	.6	2	1.7	—	—	—	—
—	—	—	—	—	—	—	—	4	1.4	4	2.3	—	—	—	—	—	—
2	.6	3	.8	1	.5	2	1.4	—	—	—	—	—	—	—	—	—	—
12	3.5	18	5.1	10	4.7	8	5.6	13	4.5	10	5.8	3	2.5	1	.8	—	—
1	.3	—	—	—	—	—	—	2	.7	2	1.2	—	—	3	2.3	2	3.7
1	.3	4	1.1	4	1.9	—	—	3	1.0	2	1.2	1	.9	—	—	—	—
—	—	—	—	—	—	—	—	3	1.0	3	1.7	—	—	1	.8	1	1.9
1	.3	—	—	—	—	—	—	1	.3	—	—	1	.9	—	—	—	—
1	.3	1	.3	1	.5	—	—	6	2.1	5	2.9	1	.9	—	—	—	—
1	.3	—	—	—	—	—	—	2	.7	1	.6	1	.9	1	.8	1	1.9
10	2.9	1	.3	1	.5	—	—	8	2.8	3	1.7	5	4.2	7	5.4	2	3.7
3	.9	1	.3	1	.5	—	—	1	.3	—	—	1	.9	2	1.5	—	—
—	—	—	—	—	—	—	—	1	.3	1	.6	—	—	—	—	—	—
—	—	1	.3	1	.5	—	—	—	—	—	—	—	—	—	—	—	—
—	—	1	.3	1	.5	—	—	—	—	—	—	—	—	—	—	—	—
4	1.2	7	2.0	5	2.4	2	1.4	7	2.4	5	2.9	2	1.7	—	—	—	—
—	—	—	—	—	—	—	—	1	.3	1	.6	—	—	1	.8	2	1.9
1	.3	3	.8	2	.9	1	.7	4	1.4	4	2.3	—	—	2	1.5	2	3.7
21	6.2	6	1.6	3	1.4	3	2.0	10	3.5	7	4.2	3	2.5	17	13.0	2	3.7
338	100.0	355	100.0	211	100.0	144	100.0	290	100.0	172	100.0	118	100.0	130	100.0	54	100.0

*Status Lymphaticus—7, Pyemia—6, Malnutrition of Idiocy—4, Pernicious Anemia—2, Septicemia—2, Diabetes Insipidus—1, Addison's Disease—1, Leukemia—1.
‡Patient died while out of institution on visit or escape. Cause of death could not be obtained.

5				6						7					
Morons .69 I. Q.				Not Mentally Defective (Not Feebleminded) .70+ I. Q.						Mental Status Unknown					
Male		Female		Total		Male		Female		Total		Male		Female	
.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
	1.9	2	2.6	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	1.9	—	—	—	—	—	—	—	—	1	1.4	—	—	1	3.3
	—	—	—	1	3.4	1	6.7	—	—	2	2.6	1	2.3	1	3.3
	24.0	12	15.0	3	10.4	3	20.0	—	—	12	16.2	10	22.7	2	6.8
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	1	1.3	—	—	—	—	—	—	—	—	—	—	—	—
	—	1	1.3	—	—	—	—	—	—	—	—	—	—	—	—
	9.2	15	19.8	4	13.9	1	6.7	3	21.5	12	16.2	8	18.1	4	13.2
	5.5	3	4.0	—	—	—	—	—	—	3	4.0	2	4.4	1	3.3
	1.9	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	1	3.4	—	—	1	7.1	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	1	1.4	—	—	1	3.3
	9.2	1	1.3	2	6.9	2	13.3	—	—	1	1.4	1	2.3	—	—
	—	1	1.3	1	3.4	—	—	1	7.1	1	1.4	—	—	1	3.3
	3.7	—	—	—	—	—	—	—	—	1	1.4	1	2.3	—	—
	1.9	3	4.0	1	3.4	1	6.7	—	—	1	1.4	1	2.3	—	—
	3.7	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	1.9	2	2.6	—	—	—	—	—	—	6	8.1	4	9.1	2	6.8
	1.9	1	1.3	1	3.4	1	6.7	—	—	3	4.0	1	2.3	2	6.8
	—	1	1.3	—	—	—	—	—	—	—	—	—	—	—	—
	5.5	4	5.3	4	13.9	1	6.7	3	21.5	1	1.4	1	2.3	—	—
	—	—	—	—	—	—	—	—	—	2	2.6	1	2.3	1	3.3
	—	—	—	—	—	—	—	—	—	2	2.6	1	2.3	1	3.3
	—	3	4.0	3	10.4	2	13.3	1	7.1	8	10.8	4	9.1	4	13.2
	7.3	2	2.6	1	3.4	1	6.7	—	—	2	2.6	1	2.3	1	3.3
	—	—	—	—	—	—	—	—	—	1	1.4	1	2.3	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	1	1.4	—	—	1	3.3
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	1	1.3	—	—	—	—	—	—	3	4.0	1	2.3	2	6.8
	3.7	1	1.3	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	1	3.4	—	—	1	7.1	—	—	—	—	—	—
	1.9	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	1	1.4	—	—	1	3.3
	1.9	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	3.7	5	6.6	2	6.9	—	—	2	14.3	1	1.4	—	—	1	3.3
	—	2	2.6	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	2	2.6	2	4.4	—	—
2	1.9	—	—	—	—	—	—	—	—	1	1.4	1	2.3	—	—
2	3.7	—	—	2	6.9	2	13.3	—	—	1	1.4	—	—	1	3.3
2	3.7	15	19.7	2	6.9	—	—	2	14.3	4	5.4	2	4.4	2	6.8
4	100.0	76	100.0	29	100.0	15	100.0	14	100.0	74	100.0	44	100.0	30	100.0

TABLE XI

CAUSES OF DEATH IN TOTAL MENTAL DEFECTIVES COMPRISING MORE THAN 1 PER CENT. OF THE TOTAL DEATHS, ARRANGED IN ORDER OF FREQUENCY, BY SEX

Causes of Death	Total	Male	Female
	Per cent.	Per cent.	Per cent.
Influenza	16.4	18.1	14.2
Tuberculosis of Lungs	14.2	13.5	15.1
Bronchopneumonia	8.7	7.3	10.3
Lobar Pneumonia	7.4	8.5	5.9
Heart and Circulatory	6.0	5.7	6.2
Epilepsy	4.3	3.7	5.0
Diarrhea & Enteritis	4.1	4.6	3.5
Tuberculosis Other Than Lungs	4.1	4.8	3.3
Measles	3.9	3.4	4.4
Other General Diseases*	3.2	2.8	3.8
Acute & Chronic Nephritis	2.5	1.9	3.2
Cerebral Hemorrhage	1.9	1.4	2.7
Malformations	1.8	2.3	1.2
General Paralysis	1.2	.9	1.5
Diseases of Spinal Cord	1.0	1.4	.6
Diphtheria	1.0	.9	1.2
Cause of Death Unknown†	4.3	2.8	6.2

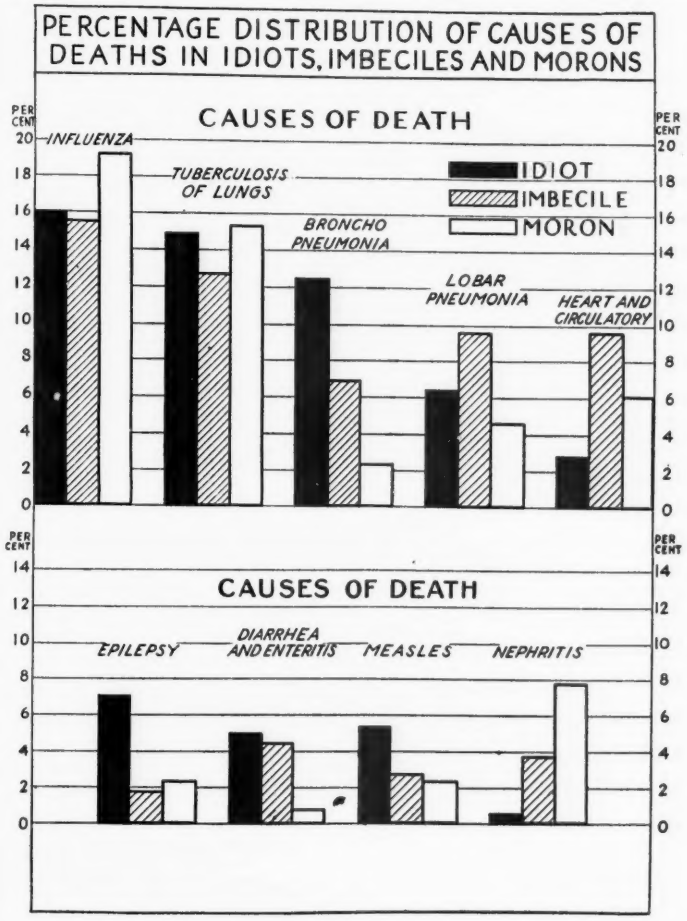
*Status lymphaticus—7, Pyemia—6, Malnutrition of Idiocy—4, Pernicious Anemia—2, Septicemia—2, Diabetes Insip.—1, Addison's Disease—1, Leukemia—1.

†Patient died while out of institution on visit or escape. Cause of death could not be obtained.

mental defectives are influenza* (16 per cent.), tuberculosis of lungs (14 per cent.), bronchopneumonia (8 per cent.), lobar pneumonia (7 per cent.), diseases of the heart (6 per cent.), epilepsy (4 per cent.), and diarrhea and enteritis (4 per cent.). The seven causes given make a total of 60.7 per cent. of all causes of death. It is interesting to group these important diseases in different ways. Thus, if we group them as infectious diseases, we note that influenza, tuberculosis of lungs and lobar pneumonia make up 37 per cent. of the total. If we group them in accordance with organs affected, we see that tuberculosis of lungs, bronchopneumonia and lobar pneumonia make up 30.1 per cent. of the total.

There are also significant differences between the sexes. If we consider the important causes we note that influenza, lobar pneumonia, and diarrhea and enteritis are more prevalent among the males. Influenza was the cause of death in 18 per cent. of males and 14 per cent. of females; lobar pneumonia in 8 per cent. of males and 5 per cent. of females; diarrhea and en-

*The high percentage in influenza may be due to the fact that the year 1918 is included in the present study, and thus the findings may not be typical. This fact should be considered as we note that influenza does not maintain first position when a comparison is made with the deaths from influenza in the general population as outlined in Table XVIII.



GRAPH G.

teritis in 4 per cent. of males and 3 per cent. of females. On the other hand, we note that tuberculosis of lungs, bronchopneumonia, epilepsy and measles play a greater part in the deaths of females. Tuberculosis of lungs was the cause of death in 13 per cent. of males and 15 per cent. of females; bronchopneumonia in 7 per cent. of males and 10 per cent. of females; epilepsy in 3 per cent. of males and 5 per cent. of females; measles in 3 per cent. of males and 4 per cent. of females; acute and chronic nephritis in 1 per cent. of males and 3 per cent. of females.

TABLE XII

CAUSES OF DEATH IN MENTAL DEFECTIVES IN MASSACHUSETTS STATE SCHOOLS, 1917-1930, BY GROUPINGS OF THE INTERNATIONAL LIST, ARRANGED IN ORDER OF IMPORTANCE

	Causes of Death	Total		Male		Female	
		No.	%	No.	%	No.	%
I.	Epidemic, Endemic and Infectious Diseases	325	42.0	187	42.8	138	40.8
V.	Diseases of Respiratory System	147	19.0	82	18.8	65	19.2
III.	Diseases of Nervous System	78	10.1	37	8.5	41	12.1
VI.	Diseases of Digestive System	63	8.1	45	10.3	18	5.3
IV.	Diseases of Circulatory System	46	5.9	25	5.7	21	6.2
II.	General Diseases	32	4.1	17	3.9	15	4.4
VII.	Non-Venereal Diseases of Genito-Urinary System	23	2.9	9	2.0	14	4.1
XI.	Malformations	14	1.8	10	2.3	4	1.2
XIV.	External Causes	11	1.4	10	2.3	1	.5
IX.	Diseases of Skin and Cellular Tissue	2	.3	2	.5	—	—
X.	Diseases of Bones	1	.1	1	.2	—	—
VIII.	The Puerperal State	—	—	—	—	—	—
XII.	Early Infancy	—	—	—	—	—	—
XIII.	Old Age	—	—	—	—	—	—
XV.	Ill-Defined Causes	33	4.3	12	2.7	21	6.2
	Total—All Causes	775	100.0	437	100.0	338	100.0

In summarizing the results for mental defectives as a whole we may say that the infectious diseases present the largest numbers of deaths. Apparently they are also susceptible to diseases affecting the respiratory system. More than this we cannot say without a comparison with deaths from these causes in the general population, which will follow later.

This table may be presented in another manner. Table XII gives us the causes of death arranged in order of importance and in accordance with the fifteen main divisions of the International List of Causes of Death. As these main divisions are arranged in order of their frequency we can gain some idea of the relative importance of the various factors. We observe that Group I, Epidemic, Endemic and Infectious Diseases, makes up 42 per cent. of all deaths; Group V, Diseases of the Respiratory System, contributes 19 per cent. of deaths; Group III, Diseases of the Nervous System, contributes 10.1 per cent. of deaths; Group VI, Diseases of the Digestive System, 8.1 per cent.; Group IV, Diseases of the Heart and Circulatory System, 5.9 per cent. The bulk of deaths comes under the first heading, that of Epidemic and Infectious Diseases. We note that the first three groups, the Epidemic, Endemic and Infectious Diseases, Diseases of the Respiratory System and Diseases of the Nervous System total 71.1 per cent. of all deaths. It is quite interesting in this connection to note the comparatively high position occupied by Diseases of the Nervous System which contribute 10 per cent. of deaths. Diseases of the Heart, which for many years have been given a high ranking as causing death in mental deficiency, is in fifth position.

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(To be Continued)

ARGUMENTS IN FAVOR OF COMPULSORY VACCINATION FOR PRIVATE SCHOOL CHILDREN*

BY SAMUEL B. WOODWARD, M.D.†

Gentlemen:

I HOLD in my hand a circular sent out by the Citizens Committee opposing compulsory vaccination. With it came a map of the United States, an enlarged copy of which appeared here last year and which I bring out again at this time.

Nine states are in black. These states have some form of compulsory vaccination.

Six states appear in stripes. Here vaccination is optional with local authorities.

Thirty-three states are in white. In these no compulsory vaccination laws exist.

*Before the Public Health Committee of the Massachusetts Legislature, February 25, 1932.

†Woodward—For record and address of author see "This Week's Issue," page 592.

Four gold-starred states have according to the circular passed all the rest in the race for freedom for they prohibit compulsory vaccination. These four states, Arizona, Utah, North Dakota and Minnesota, with a combined population in 1920 of 3,817,555 persons accumulated in their race for freedom in the ten years 1919-1928 46,130 cases of smallpox, while Massachusetts with a population slightly larger than their combined total, i.e., 3,852,356, had but 408. The free states had in other words 113 times as much smallpox as did we, and Utah taken by itself 272 times as much. Furthermore, the four states in question, Utah, Arizona, North Dakota and Minnesota had more than twice as many cases of smallpox as all the black states combined with their 29,472,362 inhabitants for these same black states had during the years in question but 20,571 cases, 13,496 of which were in the two states of South Carolina and Kentucky. Fifteen times as much smallpox man for man seems to me a rather high price to pay for this boasted freedom.

The local option states, North Carolina, Georgia, Oregon, New Jersey, Ohio and Connecticut with a population of 16,534,260 had 91,985 cases, while the no compulsory law states with their 57,636,032 inhabitants harbored the remaining 401,045 cases of smallpox. How, under these circumstances, does the Citizens Committee dare to say that wherever compulsory vaccination has been abandoned and sanitation installed in its place, smallpox has been wiped out? Let me incidentally point out that New Hampshire black, sandwiched in between the white states of Vermont and Maine, had 86 cases for its 443,003 inhabitants, while its sister states had 1,695 for their 1,130,442, or more than 8 times as many per capita and that the black state of Pennsylvania with 1,602 cases is in a direct line with the white Ohio with 37,981 cases, Indiana with 36,593 and Illinois with 31,930, 106,504 collectively, and not one of these states has the population of Pennsylvania, the three of them together but 15,175,064 to her 8,720,017. The comparison here is almost that of comparing the infinitesimal with the infinite.

I might point out that even South Carolina, designated in black on the map, with its altogether too many cases is bordered by no state with so few—North Carolina shaded, having 24,037, Tennessee white 11,000 and Georgia white 12,336 and lastly to come once more to Utah that this state with less than half a million inhabitants had during the 10 years previously spoken of 4,000 more cases of smallpox than all New England with New York, Pennsylvania, New Jersey and Maryland thrown in for good measure, with a combined population of 31,111,-714,—13,000 cases for the 434,000 people in Utah, 8,000 for the 31,000,000 of the states just mentioned.

I am more than pleased to see this map, for it has never been my good fortune to see a stronger argument for compulsory vaccination.

I come before you this year to present the endorsement of 52 private schools, four state normal schools and the presidents of 17 colleges and universities to the petition presented to you last year, and indeed for several years, to extend our school vaccination law to cover private as well as public schools.

Twelve of the schools that would be affected, tired of waiting for legislative action, have during these last years protected themselves by requiring vaccination against smallpox as a prerequisite to admission to their halls. These twelve are Cushing Academy, Middlesex School of Concord, Walnut Hill School at Natick, Munson, Bradford and Phillips Andover Academies, the Groton School at Groton, Browne and Nichols, Cambridge, Deerfield Academy, Lasell, Northampton School for Girls and St. Mark's at Southboro.

It may interest you to know that no unvaccinated pupils are now admitted to Harvard University, to Wheaton, Simmons, Radcliffe, Wellesley, Smith, Mt. Holyoke or Amherst, to Brown, Yale, Wesleyan, Cornell, Dartmouth, Vassar, Syracuse, Rutgers, Berea or Tuskegee, to Hampton, Stanford, University of Pennsylvania, University of California or the Alabama Polytechnic, or to the University of Texas or the United States Military and Naval Academies.

Educational institutions are tired of partial or complete closure under quarantine as a result of smallpox invasion and additions to the number thus protecting themselves are being made every year.

The parochial school authorities are in favor of such protection as witness letters from the Cardinal and the other two Roman Catholic Bishops of Massachusetts, and that protection which those who are particularly interested feel should be given them ought, we claim, to be provided.

There is absolutely no argument for compulsory vaccination of children in public schools and a refusal to provide the same protection for those attending private and parochial schools. In parenthesis may I say that the City of New York, which vaccinates all pupils, teachers, janitors, etc., in all schools, had in the ten years 1921-1930 but 104 cases of smallpox and no deaths and, lest it be again said that only public school children are to be vaccinated under the law, the State Supreme Court has ruled that the word school in the statute means any school, public or private.

I do not wish to take up too much of your time this morning, but as you will probably hear that vaccination is of no use, is a delusion and a sham, at least that is what I have heard yearly in this hall, I wish to tell you briefly what

Massachusetts endured before Benjamin Waterhouse of Cambridge performed the first vaccination on this side of the Atlantic and how he proved the efficacy of the practice 130 years ago last July.

In the 170 years before vaccination Massachusetts suffered from 20 distinct epidemics of smallpox, more than one every ten years. Some of these, most of them in fact, lasted more than twelve months and in the intervals sporadic cases were almost always present. No one now living in a civilized country has any idea of what a prevaccination smallpox epidemic meant. So important a thing was smallpox in the life of the community that when Captain John Bonner printed his map of the Town of Boston in 1722, the only important items listed in the margin were first "Great Fires" and second "General Smallpox."

In 1721 there were in Boston 5,759 cases of smallpox with 844 deaths and there were but 12,000 persons then living there. In other words, one person in every two had the disease and one in every 14 died of it in that year. In 1697 it was still worse, for of the 8,000 inhabitants over 1,000 died, one in eight, which in 1930 would mean the death in one year of 250,000 people of one disease in the metropolitan district of approximately 2,000,000 people. Small wonder is it that when it was discovered that inoculated smallpox was less deadly than the disease acquired in the natural way that this practice become common and people went in squads to the inoculating hospitals to take it when they felt like it, knowing that they were very sure to fall victims some time during their lives.

In 1800 Waterhouse, obtaining vaccine from England, vaccinated seven persons in his own family, and two months later sent them to the smallpox hospital in Brookline where they were exposed to smallpox by contact for two weeks and were also inoculated with smallpox virus taken from a patient then in the hospital. Not one of them had smallpox and Waterhouse well said, "One fact is worth a thousand arguments."

Two years later by arrangement with the Boston Board of Health 21 children were sent to the smallpox hospital on Noddles Island. Nineteen of them had been vaccinated, two had not. All were exposed for three weeks to infection by contact and all were inoculated with smallpox virus. None of the 19 developed smallpox. Both of the others did and then to make sure the 19 were again inoculated with virus taken from the two others, again with no result. There was no more to be said and there is no more now. As superproof, this same thing was done with 12 children in the town of Milton in 1809 with the identical result.

Smallpox at once began to lose its power and as a matter of fact in the 130 years since that time there have been but two general outbreaks, one in 1872, just 72 years later, and one in 1902, 28 years ago. We still have it because vaccination is not universal, because infant vaccination is not sufficiently general, because our neighbors send it in to us every now and then, but we could certainly do as Sweden does, as Germany does, as Paris did last year, as the Philippines and Porto Rico and Samoa and Hawaii and Guam have done, absolutely abolish the curse were we sufficiently intelligent to use the means which will abolish it, i.e., universal vaccination and revaccination.

What Turkey is doing we can do, what Russia is doing we can do and we need no longer in these United States hang our heads in shame because India and China are the only countries in the world that have more smallpox than we.

Do not be misled by the mildness of the disease in recent years here. A shift from the mild to the deadly form is always possible and such shifts occur without warning. On the fifth of December, 1921, a prisoner in jail at Poteau, Oklahoma, developed mild smallpox and recovered, but he infected 38 others with so deadly a type of the disease that 24 died. There is, and never has been, any warning in any community of this change in type.

Do we wish to have 2,567 cases in two months as Los Angeles had in 1924 and this after the health officer himself wrote me that there was no epidemic in the offing and but 60 cases in the city?

Do not emulate, in response to those who would make vaccination voluntary, the example of Gloucester, England, where the Guardians at the instance of the Anti-vaccination Society founded in 1885 gave instructions that no steps be taken to enforce the then compulsory law, for 11 years later in 1896 smallpox struck the town and struck hard with 2,035 cases and 443 deaths, a death rate of course of one in five.

Conscientious objectors are no longer vaccinated in England. In 1918 there were 63 cases of smallpox in the whole island. In 1928 there were 12,420.

In this same year, 1918, there were 16,147 deaths from 47,365 cases of smallpox in the Philippines, in 1923 four, in 1924 one, in 1925 one, in 1926 six and in neither 1927 nor 1928 any at all.

I am not here to advocate infant vaccination, but simply to ask that the State should provide for the children in the private and parochial schools the same protection now given to those attending the public schools.

HISTORY OF THE MASSACHUSETTS MEDICAL BENEVOLENT SOCIETY*

[A Personal Sketch]

BY ROBERT M. GREEN, M.D.†

MY own associations with the Massachusetts Medical Benevolent Society have always been extremely personal, and I hope, therefore, that I may be pardoned if my sketch of its history is likewise personal in its nature.

It was on a snowy evening in December, very nearly seventy-five years ago, that a group of members of the Boston Medical Book Club gathered at the home of one of their number for a regular meeting of the Club. I know not whether it were the contrast of the outdoor storm with the cosy comfort within, or that the community may have been passing through a period of depression similar to that in which we are now involved, but in any event, the conversation turned to the subject of relief for distressed physicians and their families. I have always liked the Victorian dignity of the resolution which was adopted at that meeting: "Whereas it sometimes happens that worthy members of the Medical Profession become reduced in circumstances—." With this resolution and with the subsequent work of the Committee, which was appointed to consider it, began the formation of the Massachusetts Medical Benevolent Society, which was duly organized in March of the following year. Among its original group of members were my grandfather, John Ware, and my great-uncle, Charles Eliot Ware, both of whom were associated with the Society for the remainder of their lives.

The first President of the Society was George Hayward who is to me only a name. I have often wondered at the association of the name George with the presidency of this Society; for of its ten Presidents, five have borne this name. I presume we might refer to our present incumbent, Dr. Sears, as George the Fifth. Early in the list of Presidents we encounter the names of Warren and Shattuck. Two Shattucks have been Presidents, George Cheyne and George Brune; and of the Warrens, three generations, Jonathan Mason, John Collins, and John, have been members and officers. David Williams Cheever, father of our Dr. Cheever, was President for ten years, almost the last of our distinguished line of elder surgeons from an age that is past, transmitting his qualifications to the age that is waiting before.

Among the Vice-Presidents, I note with a special interest the name of Richard Manning Hodges, and among the early Trustees that of Francis Minot. The personalities of Dr. Hodges and Dr. Minot are among my early childhood recollections; for they both had their offices in the same block on Marlborough Street as my father's home and I used to see them daily coming and going on their professional rounds. I remember, particularly, the florid face and fine aristocratic manner of Dr. Hodges. Dr. Minot used to take care of me with childhood ailments in the years before Dr. Rotch, and I can particularly well remember his attendance on my first attack of measles. Dr. Minot was Treasurer of the Massachusetts Medical Benevolent Society from 1859-1891. Indeed, the Treasurers of this Society seem to have enjoyed notable longevity for there have been only three from its foundation to the present time. Among the early Secretaries may be noted the name of Calvin Ellis, first demonstrator of the celebrated S. Curve of Ellis in pleuritic effusion.

The list of Trustees of the Society presents many names of remembrance; fourth among them is that of Morrill Wyman. His family lot in Mount Auburn is next to that of my grandfather, and when I went with my mother on Decoration Day, I can remember seeing always the figure of Dr. Wyman, who lived to an advanced age, and came always to Mount Auburn on that day. Another name is that of Charles Edward Buckingham, former Professor of Obstetrics, whose house I visited occasionally as a boy with my father. I recall, particularly, the quiet, courteous personality of Dr. Buckingham and the scholarly atmosphere which pervaded his study. Luther Parks, who was a Trustee from 1863 to 1866 and several times thereafter, was a friend of my mother's, a gentleman of the old school. I remember once meeting him when I was walking with her up Beacon Hill. On that same walk we saw Dr. Oliver Wendell Holmes and I was told to remember that I had met two distinguished men that day. The two Bigelows, Henry and Henry Jacob, served as Trustees of the Society and their names in our roster remind us of the lithotrite and the iliofemoral ligament.

With these personalities, and with many others who succeeded them, the history of the Society has been associated and its significance and strength, as with all organizations, has been dependent on the men who constituted it. Of its benefactions and work of relief, our new treasurer will presently give us some account. The Society has always been a familial affair, as things in Boston have such a way of being. This seems singularly appropriate to the nature of the work which it has done. No modern profession is so esoteric as that of Medicine. To a considerable extent, we recruit our numbers

*Read at the Annual Meeting of the Massachusetts Medical Benevolent Society, October 22, 1931.

†Green—For record and address of author see "This Week's Issue," page 592.

from the families of those already within the professional fold, and the care of those of our number who are in distress is our peculiar obligation and privilege. There is no greater compliment, which one doctor can pay another, than to request his professional care for members of his family. So it is to their professional colleagues that doctors or their families when "reduced in circumstances" can turn for aid and comfort, with assurance of kind consideration. This is the duty which our Society has been quietly discharging for three-quarters of a century and which, I believe, it will continue to perform so long as there is in our profession a thoughtful regard for those of its members upon whom misfortune and adversity have fallen.

MASSACHUSETTS MEDICAL BENEVOLENT SOCIETY*

Whom and How We Help

BY ROGER I. LEE, M.D.†

WHEN I readily agreed to tell something of the activities of the Massachusetts Medical Benevolent Society, I anticipated looking over the records and making up an interesting story from a mass of human documents. When I investigated, however, the records which my illustrious predecessor, Dr. William L. Richardson, kept, I found a very accurate account of the financial status of the society, a very accurate list of the addresses of the beneficiaries, and nothing else. As you all probably know, Dr. Richardson to all intents and purposes ran the society for a number of years. He was unalterably opposed to anything that savored of publicity. He believed that the doctors who had to be helped for one reason or another should be aided quietly, sympathetically, and privately, and the same was true of the wives and dependents of the doctors to whom the society has extended aid.

At the present time the society is helping eighteen individuals. There are two more cases pending, and very likely, in view of the hard times, there will be other applications within a short time. These eighteen individuals receive, for the most part, one hundred dollars quarterly, that is, four hundred dollars a year. No one receives more that that; a few receive less than that. In going over the reports it is evident that the majority of the beneficiaries in 1904 received twenty-five dollars quarterly. This has been slowly increased at different times, the last increase being 1929-1930. The society just about expends its income and if there are many more beneficiaries, there will have to be more income to the society or less income to each beneficiary. It is apparently a guarantee of long life to become a beneficiary of the Massachusetts Medical Benevolent Society because, of the eighteen, two have been on the list since the 90's and three have been on the list for over twenty years. It is of some interest that, of the present beneficiaries, four are physicians, one of whom is a woman physician. Of the two beneficiaries whose cases are pending, one is a woman physician. All of the other beneficiaries are women, making a total of fifteen women beneficiaries out of eighteen, of whom one, as I said before, is a woman physician.

One of our beneficiaries lives in Germany, her original home, because she finds it cheaper than living in America. Another lives in England. All of the others live in Massachusetts and are fairly well scattered throughout the state.

It is clear from a perusal of the list of past beneficiaries that eminence in the medical profession brings no immunity against need. In the list of beneficiaries kept so confidentially by Dr. Richardson are the names of eminent professors in distinguished medical schools, the bearers of names renowned in the practice of medicine, specialists, scientists, and general practitioners, and the widows and dependents of those who have had charge of our great hospitals.

If there is one impression which is stronger than any other in looking over the meagre records and the list of beneficiaries and in recollections of various conversations with Dr. Richardson and in the applications now pending, it is that the benefactions of this small society are carried out in a most sympathetic and humane fashion. If it should come to the lot of any of us, or any of our families, that hard times should fall upon us, it would not be irksome for us or them to receive the small stipend dispensed by this society, and it seems to me that this policy of quiet and kindly benefaction should be continued and perhaps extended, if there is need, but always in the simple fashion so punctiliously carried out by Dr. William L. Richardson. And I hope that it will be impossible to say that any person can be pointed out, because it is of common knowledge, that he or she receives a little help from this society.

*Read before the Annual Meeting of the Massachusetts Medical Benevolent Society, October 22, 1931.

†Lee—For record and address of author see "This Week's Issue," page 592.

MEDICAL PROGRESS

PROGRESS IN GYNECOLOGY

BY FRANK A. PEMBERTON, M.D.*

THE physiology of the ovary is the topic of interest in gynecology at the present time. In previous articles on Progress in Gynecology we have reviewed the literature on the ovarian hormones which we will again cite briefly from Johnstone[1] and others.

The reproductive cycle begins at the end of menstruation with the gradual maturation of the Graafian follicle which secretes a hormone called Estrin. Estrin is formed by all the developing follicles and probably the ovarian substance as well. It causes congestion of the genital tract and is a growth-promoting substance[2]. It seems to have some effect on the breasts as Kunde et al[3] injected it into two normal and two castrated dogs who all showed enlargement of the breasts and one of whom had such copious lactation that she nursed two puppies for two weeks. The uterine wall from one showed hypertrophy of the endometrium.

After ovulation takes place at the middle of the cycle of 28 days[4] a corpus luteum is formed which produces another hormone called Progestin. This internal secretion stimulates and governs the preparation of the uterus for nidation and gestation of the ovum, causing the thickening of and secretion in the glands of the endometrium, the so-called premenstrual phase of the menstrual cycle. It would be more correct to call it the pre-nidation phase in our opinion. If fertilization of the ovum does not occur the amount of progestin secreted is exhausted relatively quickly and menstruation occurs. It is known that if fertilization and nidation do occur the corpus luteum persists much longer than if menstruation comes on and also that the removal of a corpus luteum is followed in 2 or 3 days by a normal flow. Therefore it is assumed, but not proved, that the fertilized ovum must secrete some hormone which acts on the ovary to preserve the corpus luteum. Hisaw and Leonard[2], in experiments on rabbits, appeared to show that estrin and progestin have to be used in succession to get the full progestational effect. Either alone does not achieve the desired result. This has already been applied clinically by Novak and others as we have said in previous reviews.

Allen et al[4] have tested various parts of the human ovary for its hormone content on castrated rats by injection of follicular fluid and by implantation of fresh corpora lutea, pieces of the follicle walls, and pieces of cortical stroma free of macroscopic follicles. Corpora lutea removed on the thirteenth to seventeenth days of the menstrual cycle seemed to have the largest content of hormone with a decrease in those of the twentieth to the twenty-second day. Follicle fluid, especially from cases of menorrhagia, and follicle walls contained a good deal. The cortical stroma seemed to have none. Corpora lutea of the first and third months of pregnancy contained a good deal but two at full term contained none. Therefore the corpus luteum does not secrete the large amounts found in the placenta, blood, and urine during the later months of pregnancy. They believe that the corpus luteum merely continues activity which is primarily follicular. They find that the removal of large follicles and recent corpora lutea results in menstruation occurring in 48 hours. Therefore menstruation is a degenerative condition due to decreased action of the hormone after it has induced a certain amount of growth in the uterus. Further evidence in favor of this is that Pratt and Allen[5] have caused a flow in castrated monkeys by the injection of ovarian hormones for a period of time, the flow occurring a few days after the medication was stopped. Frank et al[7] believe that the corpus luteum secretes estrin and also progestin. Estrin has also been demonstrated in the placenta, amniotic fluid, and in the blood and urine during pregnancy[9].

Constant attempts are being made to isolate and determine the chemical composition of the ovarian hormone. Doisy et al[16] have isolated a substance in crystalline form from the urine of pregnant women which, when tested in rats, gives a reaction similar to that of estrin. They have not discovered its chemical composition. They later report[17] another substance with crystals of a different shape but having similar properties. They think it may have been changed in some way during its passage through the body but that it is derived from the former substance.

It is now believed that the anterior lobe of the pituitary gland has an effect on the ovary through the formation of internal secretions.

Wiesner[8] finds that the estrual cycle does not take place in animals from whom the pituitary has been removed but this can be remedied by implanting grafts of the gland. Crew and Wiesner[10] have demonstrated two hormones from the pituitary, one of which stimulates the ovary to form estrin and one which stimulates the formation of lutein tissue and progestin. Fresh intramuscular implantations of anterior pituitary tissue produce intense stimulation of the

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ovaries of immature rats with rapid growth of follicles and premature puberty with all the phenomena of estrus[9]. Other experiments have shown that the pituitary secretions do not have any effect if the ovaries are absent[1]. That is, the pituitary must act through the medium of the ovaries. On the other hand estrin injected into castrated animals, monkeys for instance, does cause uterine congestion[1]. Further progestin given hypodermically to castrated animals causes the changes normally found before pregnancy.

Zondek[12] found that one pituitary hormone, which he calls Prolan A, causes ripening of the follicles in the ovary and is excreted in the urine at all times, even after oöphorectomy and the menopause, but is recovered in the greatest amounts during pregnancy. He found that the other pituitary hormone, Prolan B, caused increased luteinization in the corpus luteum after the follicle ruptured and that this secretion was found in enormous quantities in the urine even early in pregnancy. Therein lies the Zondek-Aschheim test for pregnancy. If urine from a pregnant woman is injected into an immature animal such as the rat, mouse or rabbit, the follicles in the ovary mature rapidly, hemorrhage occurs into them and lutein tissue forms. This change appears in about three days and may be positive within two weeks after pregnancy has occurred. Therefore it is a very valuable test for the diagnosis of early pregnancy. Errors in the test amount to not more than 2 per cent. and may be due to the presence of new growths, especially cancer, and pelvic inflammation. Further the test is positive in chorio-epithelioma which otherwise is very difficult to diagnose, hydatid mole, and tubal pregnancy.

It is evident that great progress has been made in evolving the mechanism of the internal secretion of the ovary and its relation to the pituitary gland but not so great an advance has come in using this knowledge clinically.

The factors involved in this treatment are the exact diagnosis of the condition present in the patient and the drugs used.

The diagnosis is difficult because there are at least two glands involved, the pituitary and ovaries, and in some cases others such as the thyroid. We know that there are three internal secretions from the pituitary, one that controls growth, one that stimulates follicle formation, and one that stimulates luteinization. We know that the pituitary controls the ovaries. We know that the ovary has two internal secretions, estrin and progestin. Therefore to make an exact diagnosis in most cases we want to know how much or little of each secretion there is from the pituitary, ovaries and thyroid. The function of the thyroid can be calculated easily but that of the others not definitely. The actual tests for the others consist of the action of the hormone in the blood or urine of the patient on lower animals—rats, mice, etc. It is a clinical rather than chemical test and while it shows the presence of the various hormones, it is difficult to estimate the amount present. This can be done by using various amounts of the blood or urine in a series of animals to find the smallest quantity which will cause estrus or luteinization and estimating the total produced by the patient in rat or mouse units from that but it is time-consuming and expensive.

Frank[15], Fluhmann[13] and others have done this to some extent and are able to say how much of each substance the patient is producing but so far no definite conclusions as to the meaning of the various findings have been crystallized. For instance Frank says that the amount of hormone produced is not necessarily an accurate index of the quantitative effects to be expected. The effect depends on (1) the amount secreted, (2) the rapidity of utilization of the hormone (3) the rate and threshold of excretion. As an example he finds large amounts of the ovarian hormones in the urine in some cases of amenorrhea, meaning that the amount of secretion may be normal but the excretion is too rapid.

There is no test at present for hypofunction of the ovary other than the clinical finding of amenorrhea which might be due, on the other hand, to decreased pituitary function or too rapid excretion of the hormone. The use of the various preparations in treatment is not scientific at present.

We may be able to find out what they do by giving them to cases which are operated on later so that genital organs can be examined after treatment. Geist has done this in a small number of cases with no definite result so far.

As for the preparations themselves, many seem to be potent when tried on laboratory animals and some are not. The results in human beings certainly do not realize our desires. They must be given large amounts apparently. For instance Siebke[19] has used up to 10,000 mouse units of estrin in ten days before a menstrual period. He thinks the normal amount needed is from 250 to 1000 during each menstrual cycle. The cost of such treatment is prohibitive in most cases. The use of hypodermic preparations gives better results than oral administration but here again the results are not often commensurate with the cost and some of the drugs like the luteinizing hormone cause a good deal of distress. Further they must be continued for a long time and many patients object strenuously to having hypodermics every day, especially when no improvement can be promised. Finally we do not know how permanent any beneficial results may be.

Estrin, the luteinizing hormone from the pituitary, and extracts of the whole anterior

pituitary are available for treatment but progestin is not as yet. Theoretically estrin should be useful when the ovarian production is insufficient as in cases of amenorrhea, if it is not due to deficient pituitary secretion; oligomenorrhea and the symptoms of the menopause. The luteinizing hormone should be used when the corpus luteum secretion is thought to be weak or lacking as in epimenorrhea, menorrhagia, and metrorrhagia but one must be on the lookout for pathologic causes such as pelvic inflammation and tumors. The extracts of the whole anterior pituitary should be useful for undeveloped genital organs because they are supposed to contain the growth factor of the pituitary.

Frank[14, 15] has for some time demonstrated the presence of the ovarian hormone in the blood and can now estimate the amount in the blood and in the urine. He finds that some cases of amenorrhea and sterility show a low excretory threshold of the hormone through the kidneys. The ovaries may be producing a normal amount but it is excreted so rapidly that the normal change in the uterus is not brought about. This may prove to be of value in diagnosis. One would expect an overproduction of the hormone in menorrhagia and metrorrhagia but he has been able to demonstrate that, in only a small percentage of cases.

Fluhmann[13] has done similar experiments using blood serum instead of urine from 319 patients with various complaints to try to find some test for pituitary function. The blood serum shows as well as urine the presence of the pituitary hormone. The test was negative in women with a normal menstrual history or when the menses were scanty, persistently delayed, or accompanied by short periods of amenorrhea. The tests were positive during pregnancy, when there was no ovarian activity such as after the menopause or castration, and in a group in the early stages of the menopause with irregular profuse periods. Apparently a total loss of ovarian function causes an increased production of the pituitary hormone.

Allen and Baker[11] find that the hormone is least effective by mouth, that subcutaneous injection is objectionable, and that it is readily absorbed from the peritoneal cavity, uterus, vagina, conjunctiva and nose. Therefore they used gelatine capsules containing estrin in the form called Amniotin (Squibb) which the patient puts in the vagina. The capsule dissolves and the hormone is absorbed as has been proved by recovering estrin from the urine. Severinghaus and Evans[18] find this method beneficial for symptoms of the menopause. It may also be used in ampoule form. Siebke[19] has used estrin in the form called Menformon for amenorrhea and delayed menstruation and reports some good results. Dodds and Robertson[20] have reëstablished menstruation in 10 out of 32 unmarried amenorrheic women while 18 out of 30 married women responded to treatment. The luteinizing hormone has been used under the name Antuitrin-S (Parke Davis) and Prolan (Winthrop) but is not on the market for general use as yet. Novak and Hurd[21] report 51 cases of profuse bleeding of the functional type with relief in 44 of them. The effect has been so rapid that it seemed impossible that it should have been due to stimulation of the lutein tissue in the ovary, which is the theoretical basis on which it is used, so Novak feels that it may have a direct effect on the unknown bleeding factor in these cases. His paper gives a good review of this subject as a whole. Others have had similar results which have not been published as yet. This therapy is not old enough from which to draw any conclusion as to how permanent it will be, but it is worth trying before resorting to surgical measures.

Keene and Kimbrough[36] state their experience in the treatment of 254 cases of fibroids, 21.4 per cent. of which were treated with radium. They give the contraindications to radium treatment which we have detailed several times in these reviews. If there is anemia and flowing is not a prominent symptom necrosis of the fibroid should be thought of. They always do a curettage, finding cancer of the fundus in 1.6 per cent. of cases. Myomectomy is advised in patients under 40 if a normal, functioning uterus can be left. If a hysterectomy is done an ovary should be conserved because 81.5 per cent. have no symptoms of the menopause and only 6.1 per cent. have severe hot flushes and nervous feelings whereas after the removal of both ovaries 26.9 per cent. have severe symptoms. Wood[37] writes on the x-ray treatment of fibroids. Its advantages are no mortality, low cost as there is no hospital bill, little loss of time and a possibility of controlling the symptoms without causing a permanent amenorrhea. It is not applicable to subserous, submucous, and otherwise complicated fibroids. He feels that the use of radium entails a chance of atresia of the cervix with pyometra and an atrophy of the endometrium from its direct effect. The article is a sane, sensible one but the reviewer does not think Wood lays enough stress on the importance of an examination under an anesthetic to localize the tumor, decide about the possibility of doing a myomectomy, and rule out tumors of the ovary and endometrium. If that is done radium may be put in at that time just as well or even better than giving x-ray later. In our experience pyometra and other complications of radium treatment are very rare when a careful diagnosis is made. Lynch[38] gives his experience in treating 683 patients with fibroids. Fifty-nine per cent. of those complaining of profuse bleeding had submucous ones.

These often appear after the patient has had a child. Cancer of the fundus was associated in 1.3 per cent. Symptoms of pain and tenderness were given only with those tumors which showed "red" degeneration or necrosis. There was a sterility incidence of 28.3 per cent. which may be due to other causes than the tumor in most cases. Fifteen per cent. showed some thyroid disturbance. He transfuses before operation if the red count is below 3,500,000 or the hemoglobin 50 per cent. or less.

Graves[39] presents an analysis of cases of menorrhagia and metrorrhagia, with and without fibroids in the uterus. The histories, operations, and pathologic examinations were compared with the idea of trying to decide the cause of the flowing. Metrorrhagia was found to be associated with the absence of or defectiveness of the corpus luteum in the ovary and the actual cause of the bleeding was areas of necrosis in the endometrium. A thickened endometrium with dilated, hypertrophied glands was present in most of these cases. He calls this type endometrial dysplasia and believes that it is caused by an excess of estrin unopposed by progestin. In favor of this was the finding of follicular cysts in the ovaries from such patients. Periodic dysfunctional bleeding is due to an imbalance between the follicle and corpus luteum hormones and endometrial dysplasia may or may not accompany it. He believes that when fibroids are present they are merely incidental and have no relation to the actual cause of the abnormal flow.

Donaldson[22] has written an excellent review of the present status of radium treatment of cancer of the uterus. Canti observed in 1923 that immediately after treatment mitoses had disappeared in pieces of tissue removed for examination. Then they reappeared reaching a maximum number on the fourth day. These mitoses however were very abnormal, showing great disorganization and the whole cells were greatly swollen. After the sixth day the mitoses disappeared and the cells became necrotic. The cancer tissue as a whole disappeared in five to six weeks. It is generally believed that if the surrounding normal tissue is over radiated its resistance to invasion by cancer is lowered and the disease may spread rapidly.

Some types of cancer seem to be more resistant to radium than others. There is no definite agreement as yet but most observers think that the more undifferentiated the tumor is, that is, the more atypically it is growing, the more sensitive it is to radiation. The round cell type is perhaps more easily killed than the spinal. The cauliflower growth is more amenable to treatment than the inverting, ulcerating type but bodily resistance may be the factor concerned. That is the tumor grows out in a mass because the tissues have some quality which prevents invasion. Many cases show a marked infection and in these the tumor is destroyed with more difficulty by the treatment but this might be due to increased resistance to radiation or perhaps chemical bodies are formed in the presence of infection which stimulate the growth.

Besides necrosis radium treatment causes endarteritis and connective tissue formation, both of which hinder further extension of the tumor. Repeated exposures seem to be accompanied by a lessening of the effect on the growth. Therefore the full amount of treatment should be given in the course of a few weeks.

Radium may be used in small amounts for several days at a time or in large amounts for a number of hours. It may be applied to the surface or imbedded in the tumor. It has been inserted from above by opening the abdomen, placing radium needles or seeds where needed, and then opening the abdomen six or seven days later to remove the needles. X-ray treatment or massive doses of radium may be added from the outside.

In regard to diagnosis Novak[23] says that most cases are recognizable macroscopically but that, when in doubt, one need not fear to do a biopsy if the wound is cauterized immediately.

Schiffman[24] says that when confronted with abnormal bleeding one should keep in mind as possible causes to be ruled out:—senile vaginitis, cancer of the vagina, cervical and fundal polyps, cancer of the cervix and fundus, cancer of the tubes, and cancer of the ovaries. The last two should always be kept in mind and if bleeding occurs after the menopause for which no other cause can be found it is logical to open the abdomen to rule them out. Horwitz[25] says that in 1237 cases of cancer of the uterus at the Mayo Clinic only ten occurred in Jewesses. At the Curie Hospital in Paris it is one-fourth as frequent among Jewesses as among Gentiles. Cancer of the breast occurs with the same frequency in both but cancer of the digestive tract is twice as frequent in Jewesses. There is no valid reason known for this.

A further lead in diagnosing cancer of the cervix early is that reported by Lahm[26]. He says that the normal squamous epithelial cells of the vagina and cervix always contain glycogen which can be demonstrated by Best's stain. The amount of glycogen in them decreases when a change to carcinoma takes place. Young epithelial cells growing over eroded areas showed none. Malignant, undifferentiated tumor cells also showed none but 11 out of 13 cases where there was good differentiation did contain some glycogen. Adenocarcinoma of the cervix and fundus contained none unless epithelial pearls were found. Contrary to this are the findings

given in an abstract of an article in which the author's name was omitted[27]. He found glycogen in 60 per cent. of the squamous—and 20 per cent. of the adenocarcinomas with an increase in tumors treated by radium so the subject is still uncertain but sounds as if something might develop of use for early diagnosis.

Jeanneney[28] reports 9 cases of cancer of the cervix which had been left in by subtotal hysterectomy among a total of 1800 cases of cancer of the cervix. He says there is no objection to leaving the stump provided one is certain that it is healthy. When cancer occurs within one year after operation it was probably there at the time of the operation. When it appears under these circumstances the prognosis is poor and radium must be used with care because of the proximity to the intestine. Branscomb[29] cites 46 cases of cancer of the cervical stump among 1804 carcinomas of the cervix. Sixteen appeared within a year after the hysterectomy and 30 after two years. Thirty-seven of them had borne children.

Heyman[30] advises radium as the best treatment for cancer of the cervix with operation reserved for the radium failures. He uses hysterectomy for cancer of the fundus except where there are constitutional contraindications. Then radium is the choice but if the uterine cavity is large the chance of cure is not so great as when the radium tubes lie near the disease.

Mikulicz-Radecki[31] treats cancer of the cervix by giving a radium treatment, following this by a vaginal hysterectomy with later a repetition of the radium, then x-ray treatment and constant social and medical supervision. For the many inoperable cases he uses radium and x-ray. Cancer of the fundus is treated by vaginal hysterectomy with removal of the adnexa, followed by x-ray. Schiffman[24] uses operation followed by x-ray for both types.

Peham[32] treats cancer of the cervix by vaginal hysterectomy. He has an operability of 59.7 per cent., a mortality of 6.9 per cent. and a curability of 28 per cent. He thinks the addition of x-ray after operation increases the curability by 15 per cent. He does not think that the removal of lymphatic glands is important. Kermauner[33] operated on 624 cases with a mortality of 11.2 per cent. and 40.4 per cent. of 5 year cures. Combined radium and operation in more advanced cases cured 26.9 per cent. Weibel[34] reports on the Wertheim operation. Of 3184 cases of cancer of the cervix seen, 1515 were inoperable, the operability ranging from 44 to 63 per cent. from year to year. Spinal anesthesia gave the best results. Complications were as follows,—injuries to bladder, rectum or ureters, 15 per cent. fistulas, 10 per cent. wound infections 12.6 per cent. The primary mortality was 13.8 per cent. but only 9 per cent. in the last 500 operations. Forty-seven per cent. of those surviving operation were well at the end of 5 years and 40 per cent. of all those operated on. Pitts and Waterman[35] used radium in a dosage of 7-10,000 milligram hours during 6 days and had a relative 5 year cure of 57.9 per cent. and an absolute of 20.6 per cent.

Conservatism in the treatment of pelvic inflammation is generally practiced now because most cases recover without operation if given the chance. Priestly and Payne[40] did this in 278 cases with the usual good result. If operation has to be done they conserve an ovary and the uterus if it seems possible because if the uterus is removed the ovary probably atrophies in a few months, and it has a good psychologic effect on the patient. The ovary becomes cystic in less than 15 per cent. and re-operation is needed in only 2.1 per cent. Siedentopf[41] has used Polak's method of causing a temporary amenorrhea to give physiologic rest in subacute cases of pelvic inflammation and reports an apparent beneficial effect in 50 per cent. of the cases. Little[42] says that some acute cases have to be operated on because of the strong possibility that they may be suffering with acute appendicitis. If this occurs and salpingitis is found, he aspirates the tube if it is swollen and injects a mixture of 10 per cent. turpentine in oil into the lumen. Up to 70 c.c. has been used. There is a profuse leucorrhoea in the next few days, the mass disappears in most cases, and the symptoms are relieved. This has been done in 200 cases of which he reports 75. Four of them became pregnant subsequently.

Newell, et al[43] have made an intensive search for ova in the tubes of 90 patients and have recovered 5 specimens. The tubes were at first irrigated with salt solution into a watch glass but later a special clamp was placed across the uterus at the internal os, preventing flow downward into the vagina, and then salt solution was injected into the uterine cavity with a hypodermic syringe. The salt solution came out through the tubes into a watch glass under each distal end and whatever came out was examined for ova. They fixed the time of ovulation as from the thirteenth to the fifteenth day after the beginning of the last period. The specimens are interesting as being the earliest free ova which have been found.

Sampson[44] has written on endometriosis occurring in the cornua of the uterus after salpingectomy. He had to do a hysterectomy in 100 patients who had had a previous salpingectomy on one or both sides. These 100 uteri presented 147 tubal stumps or resected cornua and careful study of them showed misplaced Mullerian tissue in 112 instances. In 3 the distal end of the stump was patent and 2 showed pregnancy in the stump. One hundred uteri with 200 intact cornua showed only 16 instances of the condition and 100 cornua from 50 cases

of salpingitis, which is a recognized cause of endometriosis in the cornua, showed only 19 instances. Therefore he feels that salpingectomy spreads endometrial seedlings in the wall of the uterus and suggests that it may also be due to over-activity of the tubal lining during the repair of the salpingectomy wound. Further he thinks it would be wise to excise the tube with the cautery rather than the knife to prevent this occurrence. He found the tumors invading the abdominal wall where a fixation had been done in three cases and growing into the intestinal wall where it had become adherent. He believes it may lead to endometriosis in the ovary and adds to the number of adhesions present. Most of the patients were re-operated on for fibroids, cancer, and prolapse and he queries, since their reproductive ability had been destroyed by the first operation, if it would not be better on the whole to show a more radical attitude when one operates for pelvic inflammation by doing a hysterectomy rather than a salpingectomy.

Te Linde[45] describes an interesting and rare tumor of the ovary called a granulosal cell tumor. Only 36 have been reported including the four he presents. Seventy per cent. occur after the age of 45, most of them after the menopause. They are usually unilateral varying from the size of a normal ovary to six inches in diameter. They are encapsulated, soft, and usually cystic. The individual cells resemble the granulosa cells lining the graafian follicle and they tend to form structures like the follicle. Mitoses are frequent in the small ones, not in the large and they are relatively benign. The most significant symptom is bleeding after the menopause and hyperplasia of the endometrium is usually found. The latter is probably a causal relationship being due to the activity of the follicle cells. The conclusion to draw is that any patient who has bleeding late in life for which no cause can be found and which shows a hypertrophied endometrium should have an exploratory abdominal, even though no tumor can be felt, because she may have a small granulosal cell tumor. It may be added parenthetically that cancer of the tube may be found in similar cases which do not show an active endometrium.

Schleyer[46] has compiled the records of 97 cases of cancer of the ovary seen in the First Women's Clinic in Vienna. It was unilateral in 52 and confined to the ovary in only 39. The mortality of operation was 12.4 per cent. Ascites was present in 21.7 per cent. and menstruation was normal in 83 per cent. He advises exploration in all cases, the removal of both ovaries in all possible instances and the prevention of rupture of the cyst during operation. Twenty-one per cent. were alive at the end of five years when the tumor was removed intact, but of 13 cases which were ruptured, only 15.4 per cent. were cured.

Caylor and Masson[47] find that of 280 fibromas of the ovary removed at the Mayo Clinic, 4 showed a sarcomatous change, 1.5 per cent. These patients are from 55 to 66 years old and two of them had postmenopausal bleeding. The tumors have ascites associated with them at times. Here is another cause for bleeding after the change of life.

Novak[48] writes on the origin of chorioepithelioma describing a case which showed no primary tumor in the uterus but extensive metastatic nodules throughout the body. He states that this disease has been found in the testis and in the ovary in 25 cases as a primary tumor. Some have been reported in dermoid cysts. It is not uncommon to have it occur after pregnancy or hydatid mole in various parts of the body with no primary growth to be found in the uterus. When this occurs either the uterus is not the point of origin or a malignant change has occurred in the emboli which normally occur during pregnancy or the primary tumor disappears. If there is a good history of pregnancy or mole the last chain of events is the most probable.

In another article[49] he discusses the occurrence of lutein cysts in the ovaries with mole or chorioepithelioma. They probably appear of some size in every such case and may rarely become enormous cysts. Spontaneous regression is the rule after the mole is discharged or removed. The cause of them is doubtful but it is known that the anterior pituitary gland causes luteinization in the ovary and pregnancy causes changes in the pituitary. It may be that a hydatid mole stimulates the pituitary to a more marked degree than normal pregnancy with the obvious response on the part of the ovary. An autopsy on one of Novak's patients corroborates this idea because the pituitary showed a more marked and persisting pregnancy reaction than usual.

Two cases of mole with lutein cysts the size of a fetal head and an orange respectively which disappeared spontaneously in two months after the curettage are reported[50].

There are a few articles on Trichomonas Vaginale which show that the ideal method of treatment has not been achieved. Furniss[51] warns against confusing this disease with senile vaginitis and says he has had the best luck by using douches of bichloride of mercury in a 1-4000 solution. In the discussion of this paper Carey remarked that 2 per cent. silver nitrate was effective and Holden reported that he dried the vagina carefully, instilled mercurochrome solution, and inserted a wool tampon covered with Lassar's paste which was removed in two days.

Wendel[52] says there is a type of dysmenor-

rhea due to what he calls interstitial thyrotoxicosis. These patients complain of dysmenorrhea, especially with pain in the right lower quadrant and also have the objective and subjective symptoms of hyperthyroidism but their metabolism tests are normal. He says that they recover in six to eighteen months if treated with potassium iodide and sedatives. He warns against doing any surgery on them.

Fullerton and Faulkner[53] have compiled the records of 1851 hysterectomies done in 13 years. Fifty-eight per cent. (1078) were complete hysterectomies done by 5 visiting men with a mortality of 4.1 per cent. after which 18 fistulas occurred. Thirty-three per cent. (609) were supracervical hysterectomies done by 20 visiting men and residents with a mortality of 4.4 per cent. and no fistulas. The lower mortality of the more difficult operation is due to the greater skill of the visiting men and the fact that the sicker patients usually have as little done to them as is possible, so many of them fall in the second classification. The writer of this review feels that the occurrence of 18 fistulas (1.6 per cent.) after the complete hysterectomies is an argument against the present widespread agitation for doing this operation in many cases simply to get rid of the cervix for fear that it will have cancer occur in it later. There are other ways to meet the situation that are better and which we have described at some length in previous reviews. They also report 164 (9 per cent.) of the vaginal hysterectomies with a mortality of 3.6 per cent.

It may be well to emphasize again the importance of suturing the broad and round ligaments to the vagina after a hysterectomy to prevent prolapse of the vagina later on. Kennedy[54] describes the method he uses in the complete abdominal operation. It involves closing the vagina, suturing the uterosacral ligaments to it, then closing the fascia around the vagina over the area, and finally bringing in the round and broad ligaments. He feels that the fascial step is very important.

Sterilization of mentally defective convicts is being carried on in some states now and the question of whether it affects the person's general health is important. Vruwink and Popenoe[55] have investigated it from the point of view of its effect on the libido. They say that one of every four American women is normally indifferent or antagonistic to coitus. When the operation involved the tubes alone one-third of the patients had an increased desire for coitus and the others said there was no change except when the operation removed a painful pathologic condition. This result may well be due to the removal of the fear of pregnancy. If the ovaries had to be removed the libido was lessened or lost in 75 per cent. This seemed to depend also on whether the patient was young, before her desire had been developed by marriage or near the menopause, when it was lessening anyway. They felt that the healthy married women in the prime of life showed no change in the libido when the ovaries were removed.

Düntzer[56] analyzed 1500 women from 15 to 35 years of age from the point of view of taking exercise during menstruation. Their conclusion was that gymnastics did no harm to the person if she was used to this type of exercise during the catamenia but that it might be well to limit it somewhat on the day before the flow began.

Frank[57] reports an unusual tumor occurring in the pelvis, a hemangioma of the pelvic connective tissue. The patient was a single woman of 35. She had been operated on for the same thing 6 years before. He found a mass rising to the umbilicus and filling the pelvis, the origin of which could not be ascertained. He got most of it out in spite of severe hemorrhage. She had a recurrence in two years. Operation revealed a hopeless situation so he treated her with radium and two years after that no sign of any tumor could be made out.

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THE EXCESS OF TRAINED NURSES

Training schools must curtail the steady production of more nurses or the morale of the nursing profession will break down completely. This is the opinion of the Committee on the Grading of Nursing Schools, following an analysis of early returns of the 1930 census on workers.

How serious the oversupply of graduate nurses has become is revealed by the Grading Committee after tabulating figures for eighteen states and the District of Columbia. For this group, since the 1920 census, the total population has increased 7 per cent, while the total number of trained nurses has increased 78 per cent.

Although there are still areas of the country and groups of patients not properly nursed, owing to poor distribution and lack of special training, the figures for the forty-two cities studied show that the average nurse has no more than 149 days of employment in any given year, according to the present sickness rate. In Bangor, Maine, she has not more than 77 days of work, and in Ottumwa, Iowa, where employment conditions are best among the cities studied, she can work not more than 201 days in the year. Rates for the other cities range between these two.

States as a whole are somewhat less oversupplied with nurses, although in New Hampshire the nurse can expect no more than 190 days of work in the year, and in Maine, Vermont, Iowa, North Dakota, South Dakota, Kansas, Delaware, Montana, Wyoming, Arizona, Nevada, Idaho and the District of Columbia there is not nearly enough nursing to be divided between the trained and untrained nurses competing for patients.

Untrained nurses are not on the increase, census figures show, but trained nurses are being turned out to terrific competition by the thousands each year. In Maine, for example, there was in 1900 one trained nurse for every 5,068 persons; in 1910, there was one for every 910; in 1920, one for every 579, and in 1930 one for every 349.

"If nursing is to avoid disaster," says Dr. May Ayres Burgess in presenting these figures in the March number of the *American Journal of Nursing*, "the steady production of more students, who become graduates, must cease. It must cease not only in the small schools, but in most of the large schools as well. Graduate nurses must be employed. If they are unemployable, they must be reëducated. The schools of nursing have produced them. They are members of the profession. Unless their morale is to break down completely, they must either be eliminated or utilized.

"This is a year of national economic distress. Hospitals are short of funds. How, then, can hospitals take care of their patients with reduced numbers of student nurses, with increased numbers of graduate nurses, and without increasing the annual budget? Let us not assume that there is no solution for this problem. Nurses who are intimately familiar with hospital administration may be able to discover new economies, new methods of organization, which will make reductions in the number of student nurses possible. Unless some solution can be found, which it is within the practical means of the hospital to adopt, nursing will continue to grow in numbers and in distress."

What the census figures will show for densely populated states, such as Massachusetts, New York, Pennsylvania, Illinois and California, no one yet knows, but nursing leaders feel grave concern. Census reports for these and other states are being analyzed by the Grading Committee as rapidly as they become available.

"Even with the overproduction of nurses, there still exists the unnursed patient," comments Miss Mary M. Roberts, R.N., editor of the *American Journal of Nursing*. "Many persons are chronically sick at home; among these are persons suffering from arthritis. New and scientific knowledge on the care of arthritis brings new opportunities in nursing. The field of psychiatric nursing, too, is hardly touched. Patients with heart disease and tuberculosis require special nursing care. Some of the unemployed nurses can well take up special training that will fit them for these types of service."—*Bulletin American Nurses' Association.*

THE ONE HUNDRED AND FIFTIETH ANNIVERSARY OF THE DISCOVERY OF TUBERCLE BACILLUS

March 24, the special day for celebrating the fiftieth anniversary of the announcement of the discovery of the tubercle bacillus by Robert Koch in 1882, will be observed in connection with the Early Diagnosis Campaign. Various associations throughout the country have made advanced arrangements for special features to commemorate the event on that date. The special translation by Dr. and Mrs. Max Pinner of Koch's original paper of March 24, 1882, with introduction by Dr. Allen K. Krause has been published and is ready for distribution. Price 50c a copy. Write your state tuberculosis or health association. Feature stories, suggestions for radio programs, a 4-page leaflet on the life of Koch, offered in the Early Diagnosis Campaign publicity kit, may be well adapted for use in widespread publicity. —*Bulletin of the National Tuberculosis Association.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 18111

COUGH AND FEVER OF THREE WEEKS' DURATION

SURGICAL DEPARTMENT

An eight year old boy of American parentage entered complaining of fever and cough of two and a half weeks' duration following tonsillectomy and adenoidectomy a month before admission.

Following the operation he did not recover his former vigor. He stayed in the house, seemed tired, lost appetite and was not inclined to play. Two weeks after the operation he had a severe chill with a temperature of 103°. The following day he had slight cough and complained of pain in the epigastrium. The cough and fever had persisted. The cough was never very severe. There was little sputum, never blood stained or foul. An x-ray is said to have shown fluid in the right chest. A chest tap just below the right scapula gave only two or three drops of blood. Since the onset the child's appetite had failed markedly and he had lost considerable weight. He passed much gas with his bowel movements.

His birth was normal. He was breast fed for nine months. Since infancy he had had recurring attacks of tonsillitis and heavy colds. He had double otitis media at three years. Both ears were drained. He had had mumps and chicken pox. The summer before admission he had a light attack of pertussis without complications.

His mother was nervous and had some rheumatism. His father's family had a history of tuberculosis. The grandfather, an uncle and also a maternal aunt were tuberculous.

Clinical examination showed a slender, somewhat undernourished, sick-looking boy with dry roughened skin. The tonsils were entirely removed. There was slight redness in the fossae. The chest was somewhat flattened. The expansion was slightly less on the right than on the left. There was some splinting on the left side. There was marked dullness from the sixth intercostal space in the right axillary line all the way round on the right side of the vertebrae. There was a clear cut pleural friction rub posteriorly from the sixth to the tenth interspaces heard at the end of inspiration only. There were fine crepitant râles and occasional musical râles from the seventh to the fifth interspaces on the right posteriorly. The left chest was negative front and back. The heart was normal. The abdominal reflexes, knee jerks and ankle jerks were normal. The joints were enlarged, and with the flat chest and winged scapulae suggested old rickets.

The amount of urine is not recorded. The specific gravity was 1.002 to 1.014. There was a trace of albumin at one of four examinations. The sediment was negative. Blood examination showed 16,100 to 37,900 white cells, 75 per cent polynuclears, hemoglobin 80 to 55 per cent, reds 3,000,000 to 4,500,000. A smear showed slight variation in size and slight achromia. A Wassermann was negative. One tuberculin test, 1 to 1000, was negative. Another, 1 to 100, was positive. The sputum was thick and purulent. Examination showed no tubercle bacilli, elastic tissue or spirochetes. There were many Gram-positive cocci in pairs and short chains and a few Gram-negative rods. The stools were negative. A blood culture showed no growth.

The temperature and pulse showed marked septic swinging except for a few days the week before death. The temperature ranged from 97.2° to 105.5°, the pulse from 88 to 170. The respirations were from 20 to 70.

X-ray examination of the chest showed a rounded area of increased density with a fluid level and a homogeneous shadow at the right base. Below this level there were four areas of smaller size and decreased density which might have been below the diaphragm and in the liver. The area of density above the diaphragm was visualized in both the anteroposterior and the lateral views.

On the second day the child complained much of pain at the right base. On the fourth day the visiting physician noted: "The patient is irritable and has a hacking unproductive cough. There is marked evidence of loss of weight. The cheeks are flushed. The lungs show distinct impairment of the left apex and only suggestive impairment with distant bronchial breathing in the right chest. There is a suggestion of bilateral Kernig and neck sign." Three days later the dullness at the right base seemed possibly to be increasing. The temperature ranged slowly downward, but the difference between the daily maximum and minimum was greatly increased over that at admission. A lumbar puncture was done in the fourth lumbar space. The findings were negative. A guinea pig was injected. On the sixteenth day chest examination suggested high pneumothorax. X-ray examination showed air and fluid occupying the right pleural cavity and displacing the heart to the left. The fluid level was at the level of the fourth rib anteriorly. By advice of a surgical consultant a tap was done in the fifth interspace in the midaxillary line. With a hori-

zontal needle air was obtained. When the needle was directed downward about 10 cubic centimeters of thick green foul smelling pus was obtained.

That day operation was done. The following day x-ray examination showed the fluid down in the costophrenic angle and the entire left lung field replaced by a shadow of air, with some displacement of the heart to the right due to rotation of the patient. On the second day after operation the temperature rose to 104.5°. Transfusion of 300 cubic centimeters of blood was done. The patient felt better. A second blood culture showed no growth. The wound continued to drain foul pus profusely however, and the chart to show septic swinging. The cough was very infrequent. X-ray examination eight days after operation showed the right lung to be about two-thirds expanded. There was still considerable air in the pleural cavity. No large amount of fluid was present. A film was requested taken in the upright position for further study of an oval area of diminished density just above the right diaphragm.

On the eleventh day after operation the visiting physician noted: "There is nothing in the heart, abdomen, joints or upper respiratory area to account for his persistent swinging temperature. The right chest still shows evidence of some fluid in the deep base with associated pulmonary infiltration. The entire left base posteriorly is dull; as one approaches the vertebral column, it is flat. This is not a pneumonic process." A third blood culture showed no growth. On the fifteenth day after operation x-ray examination showed the area of density in the left pleural space to be about the size of a drainage tube. There was marked lateral curvature of the vertebrae. There were still about ten small areas of density in the right cardiophrenic angle. A diagnostic tap posteriorly the following day showed pus.

On the seventeenth day after the first operation a second operation was done. The following day the temperature was lower. There was profuse drainage from both wounds. For the next two days postural drainage gave a little pus each time. The temperature was elevated again. There was less staining on the dressings. On the sixth day after operation 275 cubic centimeters of blood was transfused. At x-ray examination there was still a large area of pneumothorax and collapse of the right lung. The areas of density were not distinctly seen. The temperature gradually came down and on the eleventh day after operation was normal, but on that day the pulse rose to 150 and the respirations to 70. On the fourteenth day after operation a third transfusion was done. The non-protein nitrogen that day was 28 milligrams. The patient was showing slight improvement. There was moderate drainage. He coughed considerably while sitting up and asked to have his posture changed every few minutes. The condition remained unchanged until the nineteenth day after operation, 52 days after admission, when he suddenly became cyanotic. He was placed in an oxygen tent, but gradually went into coma and died.

Clinical Discussion

BY RICHARD C. CABOT, M.D., GEORGE W. HOLMES, M.D., AND MAURICE FREMONT-SMITH, M.D.

NOTES ON THE HISTORY

Dr. Cabot: Of course that opening sentence makes us think of pulmonary abscess, because that so often follows tonsillectomy.

Of course tuberculosis is possible in this child, though at eight years one would say it is distinctly rare.

The past history seems to be of no significance.

NOTES ON THE PHYSICAL EXAMINATION

That is a diagnosis of abscess of the lung without any question at all. The fluid level in the x-ray and the physical signs are characteristic. Temperature, leukocytosis, one chill,—I do not see how anybody could ask for any more evidence than that. There may be something below the diaphragm too. As yet we have not much to back up that guess.

The visiting physician found distinct impairment at the left apex and only suggestive impairment in the right chest. Yet the x-ray of course shows trouble in the right chest.

Dr. Holmes: The first films show a triangular area of dullness at the base of the right lung between the heart and the diaphragm. In the lateral view it is shown to be in the region of the lower lobe. It may represent the whole lobe contracted or it may be an area within the lobe. In one part there are several areas of diminished density. Some of them have what look like fluid levels, so they may represent multiple abscesses. The diaphragm on this side is high, as though there were some interference with the passage of air into the lung.

In the next film we have a hydropneumothorax with collapse of the lung and a definite fluid level.

These last films are portable, showing an enormous amount of magnification. Whether that is all merely magnification or whether there are some changes in the pleura it is impossible to tell.

From these films one would be led to suspect there was a possible pericardial effusion.

This time we have hydropneumothorax, a collapsed lung and effusion.

Dr. Cabot: The lumbar puncture was done on account of the Kernig and neck sign, which did not seem afterwards to amount to anything.

When they did the chest tap and obtained thick green foul smelling pus they were I suppose in the abscess. We do not know certainly the origin of that abscess. It may conceivably have been a cavity of pulmonary tuberculosis or more probably an abscess from sepsis. Those are the two things still in our minds to be distinguished.

I should say that operation was done for abscess of the lung, presumably not believed to be tuberculous.

DR. FREMONT-SMITH: Why not for an empyema?

DR. CABOT: That is perfectly proper, but I think there is evidence of something inside the lung before the empyema or pneumothorax came. Possibly they are going into the pleura and not further.

PREOPERATIVE DIAGNOSIS, FIRST OPERATION

Pneumothorax.

FIRST OPERATION

Under local anesthesia a portion of the seventh right rib in the midaxillary line was removed. The chest was full of pus and air. No attempt was made to locate a lung abscess. One large tube and two Dakin's tubes were inserted for drainage.

BACTERIOLOGIC REPORT

One culture from the pleural fluid showed no growth. Another showed staphylococcus albus.

PREOPERATIVE DIAGNOSIS, SECOND OPERATION

Lung abscess.
Empyema.

SECOND OPERATION

Under local anesthesia a portion of the tenth right rib was removed in the posterior scapular line. The empyema cavity was entered on incising the thickened pleura. The lung was adherent just posterior to the rib which was resected. Aspiration with a needle at this point gave thick pus. An incision was then made into a small abscess cavity which seemed to be in the lung. Two large tubes were inserted for drainage.

FURTHER DISCUSSION

DIFFERENTIAL DIAGNOSIS

DR. CABOT: The precise nature of that final event I do not know.

Essentially this is a case of sepsis. I have no doubt that this history started with the tonsil operation, followed by a pulmonary abscess that broke into the pleural cavity with pyopneumothorax. They drained it, and drained it again, and the second time thought that they had got into the pulmonary abscess. They did not know; and if they could not tell I do not see how we can. He died of it however. This is the history, then, essentially of a death from sepsis. What terminal event complicated that sepsis I do not know. I can imagine pulmonary embolism, but we have no good source for an embolus, so I think we can do no more than guess.

A STUDENT: I should like to ask what happened to that left lung after operation and why. Why was that drainage tube put in?

DR. CABOT: I suppose some air got in on the left at the time of operation and later was absorbed. The serious part seems to be wholly on the right side.

DR. FREMONT-SMITH: We have no evidence for subdiaphragmatic abscess, have we? No general symptoms?

DR. CABOT: Sepsis might have gone through to the diaphragm from that description. One thinks of it generally as coming from below. My belief is that there will be found the remains of a pyopneumothorax on the right and presumably some other foci of sepsis, I do not know where. I think it will be shown that the whole thing started as a pulmonary abscess. The immediate cause of death I do not know.

X-RAY INTERPRETATIONS

The findings are now suggestive of lung abscesses with probable collapse of the lung and pneumothorax.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Lung abscess with empyema.
Pyopneumothorax.

DR. RICHARD C. CABOT'S DIAGNOSES

Pulmonary abscess.
Pyopneumothorax.
Probably sepsis elsewhere.

ANATOMIC DIAGNOSES

1. *Primary lesion.*

Multiple abscesses of the lung.

2. *Secondary or terminal lesions.*

Pyopneumothorax.
Fibrinous pericarditis.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: We found a very obvious pyopneumothorax. We also found an abscess of the lung which communicated with it. Whether it was the original abscess which broke into the pleural cavity, thus giving rise to the pneumothorax, or the one which the surgeons went into at the second operation we were not able to tell. There were also a great many other abscess cavities, as is so commonly

the case. The prognosis surgically in these cases depends to a large extent on the question as to whether the abscess is single or multiple. In many cases they are multiple, which makes it virtually impossible to drain them successfully.

The complications of lung abscess run along fairly standard lines. Pyopneumothorax is always one of the things that may happen. It is not at all rare, particularly when that has happened, for the process to extend to the pericardium, as was the case here. There was a definite fibrinous pericarditis. When cases are a little more chronic, endocarditis is not rare.

Of course one of the things one always is in fear of is metastatic brain abscess. I think they probably thought of that in this case during the short period of cerebral symptoms. If there was any such metastasis in this case it must have been a very small one, since the symptoms were so transitory.

We did not find any adequate explanation for the suddenness of the terminal event.

The peritoneal cavity was all essentially negative.

Bacteriologic examination of the lungs showed a great variety of organisms, as is usually the case. We found pneumococci of two varieties, other kinds of Gram-positive cocci, Gram-negative bacilli and Gram-negative cocci, while in the sections were some granules resembling to a certain extent actinomycosis but failing to show Gram-positive filaments with the Gram stain. It is not extremely rare in lung abscess to get peculiar little accumulations which look much like sulphur granules and show a radiate structure microscopically but which do not stain like actinomycosis. I am unable to explain their origin, but doubt if they are microorganisms at all. I have seen them also in a liver abscess in which only hemolytic streptococci were present on culture. It is very important to do a confirmatory Gram stain before making a diagnosis of actinomycosis.

DR. CABOT: Was there nothing in the liver or below the diaphragm anywhere?

DR. MALLORY: No, nothing.

CASE 18112

WHAT IS THE CAUSE OF CARDIAC FAILURE?

MEDICAL DEPARTMENT

First admission. An American station agent fifty years old entered four years and a half before his final admission. He came for treatment of a septic left knee and an abscess of the left thigh following trauma. At that time no abnormality of the heart was noted. The blood pressure was 110/60. A Wassermann test was negative. Under local anesthesia the abscess beneath the quadriceps extensor was opened and a great deal of pus drained. The sinus above seemed to connect with the sinus below the knee. X-ray showed some definite joint destruction. The left chest was smaller and less radiant than the right. The diaphragm was high on the left side. The intercostal spaces were narrow on the left. The heart and mediastinal contents were displaced to the left. There were signs consistent with pneumonia, which however would not account for the high position of the diaphragm. A flexion deformity of the knee joint developed, which was however successfully treated.

A year after discharge it was noted that amputation, which had been supposed to be inevitable, had been avoided and he now had a very useful leg.

Further history. When he reëntered, three years and three months after this note was made, he said that six months before readmission he had a severe cold followed by a cough with considerable yellowish sputum which lasted a month. He then found that he became short of breath on moderate exertion. The cough improved, but the dyspnea became increasingly severe. Two months before readmission he noticed edema of the ankles at night, especially of the left. Two weeks later he began to be wakened about twice each night by a severe choking sensation and had to get out of bed and stand near the window in order to breathe more easily. These attacks lasted about five months. A month before readmission his abdomen began to swell.

He gave a past history of typical attacks of renal colic followed by the passage of gravel about once in three years for fifteen years. On several occasions he had had hematuria lasting for a day or two. The last stone was passed a month and a half before readmission. There was no history of acute rheumatic fever.

Second admission, four years and four months after his previous discharge.

Clinical examination showed a well nourished man in respiratory distress. There was marked cyanosis of the lips and fingers and marked orthopnea. The teeth were poor. The lungs showed evidence of a considerable amount of fluid, more on the right, rising in the axilla. There was some tympany or increased resonance above the fluid on the right. There were numerous moist râles. The apex impulse of the heart was seen and felt in the fifth space. The left border of dullness was 15 centimeters to the left of midsternum, 6.5 centimeters outside the midclavicular line. The right border was 4 centimeters to the right. The supracardiac dullness was 4. There was gallop rhythm. The rate was rapid. There was a harsh systolic murmur at the apex. The blood pressure was 110/90 at entry, later 140/90 to a terminal 100/90. An electrocardiogram showed regular tachycardia, rate 125, question auricular-ventricular nodal or ventricular paroxysm, the P waves poorly

marked, occurred irregularly and slower than ventricular, low voltage, high origin T_2 and T_3. The liver dullness extended to three finger-breadths below the costal margin. The edge was not felt. There was some tenderness in the right upper quadrant. There was considerable edema of the legs, ankles, sacrum and abdominal wall. The pupils and reflexes were normal.

The amount of urine was 30 to 75 ounces, the specific gravity 1.015 to 1.025. There was a large trace to a very slight trace of albumin at all of eight examinations, 1 to 4 white blood cells per field at six, rare red cells to great numbers at all, hyalin and granular casts at three. The renal function was 10 to 15 per cent. Blood examination showed 6,040 to 10,720 white cells, 65 per cent polynuclears, hemoglobin 90 per cent, reds 5,000,000, smear normal. A Hinton test was negative. The non-protein nitrogen was 25.

The temperature was 96.3° to 102.5°, the pulse 79 to 149, with many dropped beats during the last week. The respirations were 20 to 45.

A chest tap the day of admission in the right ninth interspace in the posterior axillary line gave 500 cubic centimeters of straw colored fluid, specific gravity 1.011, 290 lymphocytes, 540 red blood cells. A culture was sterile. A guinea pig test was negative for tuberculosis. The following day an abdominal tap between the umbilicus and the symphysis gave two quarts of straw colored fluid, specific gravity 1.015, 4,100 red cells, 342 white cells, all lymphocytes.

The pulse remained rapid in spite of digitalis. The patient had less respiratory distress. Theocin gave mild diuresis. A week after the taps there was still much fluid in the chest and the abdomen. He still had moderate respiratory distress. Salyrgan was started and another chest tap was done. 400 cubic centimeters of fluid was obtained, specific gravity 1.015, 2640 lymphocytes, 560 polynuclears, 800 red cells. The patient was more comfortable but responded poorly to digitalis. A slight toxic rash appeared over the chest, armpits and groins, fading in two days. At the end of two weeks he was irrational at night and had little diuresis. For the next two weeks there was no change. Then one night he was irrational and appeared moribund. He improved temporarily, was less irrational, but showed frequent dropped beats and extrasystoles. He grew steadily worse, became completely maniacal, and a month after admission died.

Clinical Discussion

BY MAURICE FREMONT-SMITH, M.D., AND TRACY B. MALLORY, M.D.

Dr. Fremont-Smith: What did he have in the lungs at the first admission? Those are the signs of collapse in that left chest. The intercostal spaces were narrow and the heart was pulled in that direction. We would like to know whether he was acutely ill or not. If he was he could get a collapse of the lung from acute infection similar to the signs of collapse following operation which in the old days were called pneumonia. On the other hand if he did not have an acute infection this chest would make us suspicious of a primary carcinoma of the bronchus. We have no clinical story here pointing to the chest. We have nothing to go by except the x-ray statements. The knee apparently was an acute traumatic infection. There is no connection between that and the chest so far as I know.

By the time of his second admission we have two facts, one that he did not have cancer of the bronchus, because if he had had that four and a half years previously he should have heard from it again before now; and the other, that he now has signs of cardiac failure. He is fifty-four.

You may remember that I told you how often a history of the passage of a kidney stone does not close the chapter, how often years later one would still find evidence of infection and possibly of other stones in the kidney.

We have no explanation for the heart failure. That past history gives information as to his renal colic, but it does not give us any help as to his heart. Why has he a failing heart at fifty-four?

It is well to call attention to the fact that there may have been something below the heart pushing it up. In pregnancy and ascites the heart is pushed upward and outward, lying less vertically in the chest.

In the report of the electrocardiogram they are trying to explain the origin of the rapid and abnormal rhythm. They thought that it might start in the auricular-ventricular node or in the ventricle. The electrocardiogram shows a block with the ventricles going at a rate of 125. The question is where that rapid rate started. However we interpret the electrocardiogram, we have a failing heart and evidences of ascites, chronic passive congestion, enlargement of the liver and so forth. That is all I get out of the history so far.

That renal function of 10 to 15 per cent is consistent with cardiac decompensation, as are the other signs, even the granular casts.

Those red cells probably got into the fluid from a scratch of the wall by the needle.

It is strange that they should have wasted a guinea pig on that fluid. I should think it most improbable that that could have been tuberculosis. In the first place we have a good reason for the presence of this fluid in heart failure. In the second place the gravity is that of a transudate rather than an exudate. Tuber-

culous chest fluids run 1.016 or above. Moreover it is very unusual to get ascites and a chest full of fluid in tuberculosis. We get one or the other, tuberculous peritonitis or tuberculous pleurisy. I do not know that I have ever seen a case of both. Have you?

DR. MALLORY: No.

DR. FREMONT-SMITH: Did they infect that chest cavity when they tapped him? Why the polynuclears? I do not know whether we could get that in chronic passive congestion. Of course there can be diapedesis with red cells. He may have diapedesis of polynuclears.

DR. MALLORY: I should think it possible that he might have had hemorrhage after the first tap and that this was a reaction to that.

DR. FREMONT-SMITH: Exactly. That is the way one would get an aseptic meningitis.

"A toxic rash appeared." Toxic in what sense? We do not get a rash from digitalis. It may even have looked like a scarlet fever rash with no fever. That sort of rash could be called a toxic rash. No one knows the toxin. That is the distribution of the rash of scarlet fever. It might have been a streptococcus rash if he had had a septicemia. He does not seem to have had it.

I should like to see what the temperature was during all that time. He may have had a septicemia. He may simply have had the maniacal episode that goes with some cases of heart failure. His temperature was 101°, 102°, 100° and 102.5°, his pulse 130 and 150 and his respirations 25 to 30. He had a septic chart. I suppose the reason that they inoculated that guinea pig was because of that very definitely septic temperature. He had had an abscess in relation to his knee. Could he have had a little area of osteomyelitis there that caused a septicemia? The history is too long. He may have had septicemia at the end, but if so it cannot explain the whole thing. He had cardiac failure for months here. I have no idea why he should have had a failing heart at fifty-four years of age. He did not have a hypertensive heart; he did not have a luetic heart. He was hardly old enough to have had an arteriosclerotic heart. He may have had subacute bacterial endocarditis. We have none of the facts either to make or to throw out such a diagnosis. How long has he been showing heart failure? The dyspnea became strikingly more severe just before his second admission. His first entry was only four and a half years before his final admission. He was dyspneic for only four or five months, so he may have had bacterial endocarditis or even a streptococcus septicemia for as long as that.

Sepsis, failing heart, fluid in the abdomen, a history of a septic knee a few years before, a history of kidney stones, no history of rheumatic fever. The only other possibility is a rheumatic heart, and that is the only thing I have not spoken of, rheumatic heart without any very definite evidence of valvular injury, and still injury enough to the muscle to cause the heart to fail at fifty-four. I am absolutely unable to decide between that and arteriosclerotic heart disease. I think the association here is (1) heart failure and (2) sepsis.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Arteriosclerotic coronary disease.
Cardiac decompensation.

DR. MAURICE FREMONT-SMITH'S DIAGNOSES

Cardiac failure, arteriosclerotic? rheumatic?
Sepsis.

ANATOMIC DIAGNOSES

1. *Primary diseases.*

Hypertrophy and dilatation of the heart (hypertensive).
Coronary sclerosis with occlusion (right circumflex).

2. *Secondary or terminal lesions.*

Chronic passive congestion.
Acute bronchitis.
Renal calculus.
Splenic infarct.
Pulmonary congestion.
Pulmonary atelectasis.

PATHOLOGIC DISCUSSION

DR. MALLORY: I do not know why they made that clinical diagnosis in the record, but it was a good guess. He did have quite marked coronary sclerosis with definite occlusion of the transverse branch of the right coronary artery. That is a rather unusual branch to show thrombosis. The descending branch of the left coronary is usually the one affected. The heart was very much hypertrophied, weighing 650 grams. The valves were all negative. There was very little arteriosclerosis elsewhere than in the coronaries.

The right kidney still contained one stone measuring 1.5 by .5 by .5 centimeters, fairly good size.

There was a small infarct of the spleen.

There was an acute bronchitis. Blood cultures were negative, so we have no adequate explanation of the long continued fever. Bronchitis was the only evidence of sepsis that could be found, but it hardly seems that that was sufficient to account for the fever.

A STUDENT: In the absence of any explanation for the failing heart, is it safe to advance a coronary?

DR. MALLORY: No.

DR. FREMONT-SMITH: I think we did have

evidence of coronary disease here, because he has abnormal T waves and auricles and ventricles not running together. There was a block, and we must have some explanation for it. We can have block from rheumatic heart disease, from coronary occlusion, from ordinary arteriosclerosis. Gummas have been reported in the region of the auriculoventricular bundle giving heart block. I think however that with a man of fifty with block we have reason to believe we have sclerosis of the coronaries.

A STUDENT: In heart block isn't it rather unusual to have a heart rate of 125?

DR. FREMONT-SMITH: Yes, unless there is a point of irritation in the ventricle giving extrasystoles in strings which might simulate paroxysmal tachycardia. We can have a point of irritation in the ventricle and get a tachycardia from that, in which case the auricle would not be going along at the same rate.

A STUDENT: But they state definitely that the tachycardia was regular.

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DR. HARVEY CUSHING ON INTRACRANIAL TUMOURS

DR. HARVEY CUSHING reported, at the International Neurological Congress held in Berne, Switzerland in the summer of 1931, on over two thousand brain tumours verified by either operation or postmortem examination. This report has now been published in the form of a monograph*, the largest series of brain tumours treated by a single individual ever to be collected. A careful analysis of this material from the pathological point of view has resulted in a number of books previously published from Dr. Cushing's clinic. The present contribution is largely statistical, but, as it contains his mature judgment in regard to the various types of brain tumours and the best operation for each, it becomes more than a list of numerical facts. So important is this work that a somewhat detailed analysis of its contents seems justified.

*Cushing, Harvey. Intracranial Tumours: Notes upon a series of two thousand verified cases with surgical-mortality percentages pertaining thereto. Charles C. Thomas, Springfield, Ill., 1932. xiv + 147 pp.

The report is on 2,023 verified tumours, divided into 11 different groups as follows:

I.	Gliomas	42.6%
II.	Pituitary adenomas	17.8%
III.	Meningiomas	13.4%
IV.	Acoustic tumours (Neurinomas)	8.7%
V.	Congenital tumours	5.6%
VI.	Metastatic and invasive tumours	4.2%
VII.	Granulomatous tumours	2.2%
VIII.	Blood-vessel tumours	2.0%
IX.	Sarcomas (primary)	0.7%
X.	Papillomas (Choroid plexus)	0.6%
XI.	Miscellaneous	2.2%

The *gliomas*, therefore, form by far the largest group. There were 687 verified cases, of which the *astrocytomas* and the *glioblastoma multiforme* tumours were 255 and 208, respectively. Gliomas vary greatly in their malignancy and, by the use of the supravital method of examination, the probable histological nature of the tumour can be quickly ascertained at the time of the operation. Many gliomas are slow growing and favorable for surgical removal. This is particularly so of the astrocytomas, more especially those occurring in the cerebellum. Dr. Cushing's case-mortality rate for these tumours is 3.4% with an operative-mortality of 2.9% and the rate for all astrocytomas is not very much higher. Radical extirpation or evacuation of these tumours, even when they lie in areas which cause paralysis and aphasia, is often advisable, as a surprising degree of restoration of lost function may occur when tension has been fully relieved. They should not be treated by decompression alone. The glioblastoma multiforme tumours are more malignant and rapidly growing. In spite of their infiltrating character, they should be attacked vigorously by a combination of block extirpation aided by suction with electrical "scalloping". When the tumour has attained a considerable size, its central portion has usually become so necrotic and broken down that it is easily removed by suction. They should be thoroughly radiated after operation. In many cases there is only a short span of six to nine months before recurrence takes place. Secondary operation must be done, but if this method is carried to its final conclusion, a 100 per cent. case-mortality must be expected.

The other types of gliomas vary greatly in their malignancy. Many are almost impossible to remove and in, for instance, the *cerebellar medulloblastomas*, the operative-mortality may be as high as 26.3%. These tumours are most susceptible to the effects of radiotherapeusis.

Pinealomas are very rare tumours; there have been practically no successful operations upon them. Dr. Cushing states that he has never succeeded in exposing a pineal tumour sufficiently well to justify an attempt to remove it and in 8 of 14 cases no operation was attempted. Six patients operated upon all died in the hospital, a case-mortality rate of 100%.

The final summary on the gliomas is as follows: "The combined mortality percentages for the 862 verified gliomas of all types, 1,173 operations having been performed at different times on 780 patients with 202 postoperative fatalities, give a 25.9 per cent. case-mortality and a 17.2 per cent. operative-mortality. The figures for the past three-year period show 282 operations on 198 new patients observed in this interval, giving a present-day 15.7 per cent. case— and an 11.0 per cent. operative-mortality for gliomas as a whole irrespective of their type and position."

The *pituitary adenomas,* representing in Dr. Cushing's clinic about 18 per cent. of all intracranial tumours, may be exposed either by the transphenoidal operation or by the unilateral transfrontal approach. Dr. Cushing has compared his figures for the two types of operation, using his records from 1921-1931, and finds that the transfrontal procedure definitely takes precedent over the other, not only in saving vision but also in the mortality rates. The general mortality rates for this group are low, somewhat less than 7 per cent., and these have been reduced in the last three years. These figures may soon approximate 1 or 2 per cent. Radiotherapeusis for pituitary adenomas is gradually being superseded by operation.

The *meningiomas,* representing about 13 per cent. of all tumours, are usually considered favorable for removal. At operation, however, they offer many difficult technical problems as they may attain a huge size before being discovered, have great vascularity, or involve one of the major venous sinuses. They are practically the only intracranial tumours for which it is desirable, routinely before operation, to prepare for the possible need of a blood transfusion and they remain today the only tumour in which one may still feel occasionally obliged to resort to the old two-stage operation. Moreover, these lesions, supposedly benign, are really very prone to recur, even with great rapidity. Occasionally there is a long interval between the first appearance and a recurrence. One patient showed signs of recurrence twelve years after an operation supposedly completely removing a meningioma and five years later the growth had become so large that a fatality occurred from bleeding after its attempted removal. In general, therefore, the mortality for these tumours is fairly high, 11.6 per cent. in Dr. Cushing's series, and the operative-mortality 7.7 per cent.

There is still considerable debate in regard to the surgical treatment of the *acoustic neurinomas.* They are benign tumours and total enucleation would be the ideal procedure. When this is attempted, however, the operative mortality is very high, formerly being 60-75 per cent. In the average case, if the pressure effects of the tumour can be so far overcome by an intracapsular excavation as to permit a subsidence of the choked disc and thus to save vision, one may well be content; and should a secondary operation for recurrence ever be necessary, it need not be particularly dreaded.

A new principle added to the technique of operation is described as cerebellar "uncapping". By this is meant removing an overlying shell of cerebellum, allowing the tumour to be effectually exposed and more easily removed. In the last 50 cases, in all but twelve of which the tumour was uncapped before removal, there were only two fatalities, giving a 4 per cent. case-mortality, compared with 18 per cent. for a series of forty-eight cases up to 1921.

Congenital tumours, particularly *craniopharyngiomas,* are often difficult to remove and the case mortality is more than 20 per cent. This also applies to *cholesteatomas, dermoids, teratomas* and *chordomas.*

Metastatic lesions and invasive tumours of the brain constitute 65 of the series; 48 were *carcinomas,* 12 *sarcomas* and 5 *hypernephromas.* Most of the metastatic tumours were stumbled on unexpectedly. Occasionally palliative operations were done for the increased intracranial pressure. A few cases of primary brain tumour were found where metastatic tumours were expected. Usually metastatic tumours are multiple. The mortality rates are necessarily very high.

Of the *granulomas, tuberculomas* are the most common, although these are very rare tumours. Tabulations of forty years ago gave tuberculomas as 30 or 40 per cent. of all intracranial tumours; now they are less than 2 per cent. Tuberculomas are much more common in the posterior fossa. When they are removed from the cerebellum, tuberculous meningitis is likely to ensue within three months. Operation is, therefore, not advised except as a means of decompression, heliotherapy being substituted. Occasionally there may be recovery. Thirty-three patients were operated upon 35 times with 15 fatalities, a 50 per cent. case-mortality. Recently the statistics have been somewhat improved.

Syphilomas are exceedingly rare, particularly since 1911. They are, moreover, particularly resistant to antisyphilitic treatment and surgical measures are indicated in the majority of cases if for no other reason than the preservation of vision. In 12 cases in the series 10 were operated upon 14 times with no fatalities. No case has been encountered in the last three-year period.

Blood-vessel tumours represent only about 2 per cent. of intracranial tumours. Many are impossible to operate upon and the case-mor-

tality is high, at least 25 per cent. They should be treated by radiation.

Sarcomata, papilloma of the choroid plexus and a few miscellaneous tumours are also reported upon.

In general, the mortality percentages have been greatly reduced in the last thirty years: *gliomas,* 30.9 to 11.0 per cent.; *pituitary adenomas,* 13.5 to 5.7 per cent.; *meningiomas,* 21.0 to 7.7 per cent.; *acoustic tumours,* 25.0 to 4.4 per cent. The reasons for this are clearly given by Dr. Cushing, who writes, "It has been erroneously assumed that the improved results of recent years are due to earlier diagnoses rather than to greater skill and experience. . . . This assumption is far from the actual facts. In reality, each year problems become more difficult than those of the year before. . . . What is more, each succeeding year sees tumours surgically exposed, like tumours of the third ventricle, which formerly were regarded as hopelessly inaccessible. Hence, all things considered and in spite of the constant improvement in diagnosis and surgical technique, the operations as time passes become increasingly critical and difficult."

The principal steps which have made it possible not only to attack the more formidable problems of the present day but at the same time to lower the operative mortality rates are as follows: the generally accepted methods of decompression to relieve tension; irreproachable wound healing; the separate closure of the galea by buried suture; the introduction of local anesthesia, occasionally supplemented by rectal anesthesia with tribromethanol; the more precise localization which, in obscure cases, Dandy's ventriculography permits; the use of motor-driven suction apparatus; the improvements in methods of hemostasis, particularly the introduction of electrosurgical devices; and better postoperative care of patients.

The report is extensively illustrated with photographs of patients and pathological specimens, as well as line drawings of operations.

CRITERIA FOR TONSILLECTOMIES

THE Department of School Hygiene of the City of Boston has recently issued an instruction letter to school physicians listing criteria for tonsillectomies, standardized in so far as it is possible in the light of our present knowledge. Family physicians are prone to resent apparent interference with their own functions in these respects and have frequently felt that they had grounds for disagreement with examining physicians of the schools in such matters. Carefully formulated rules, such as those which have been issued by Dr. Rubin, should tend to bring into better accord the school and family physicians, especially since it is emphasized that the school physician is acting merely in an advisory capacity and the final decision rests with the family physician.

According to these rules, which any fair-minded physician is bound to accept as adequate, the school physician should recommend tonsillectomy and adenoidectomy only:

1. If the child gives a history of repeated attacks of acute tonsillitis or quinsy sore throat;
2. If the child has suffered from continuous head colds;
3. If the cervical glands are definitely enlarged and have suffered from acute inflammation;
4. If the child has obstructive nasal breathing (adenoids);
5. If the child has suffered from an attack of acute catarrhal or suppurative otitis media;
6. If the child is a mouth breather;
7. If the child gives a history of repeated attacks of pharyngitis, laryngitis, or bronchitis;
8. If the tonsils obstruct the fauces, making the swallowing of food difficult;
9. If the child gives a history of rheumatism, chorea, endocarditis, or nephritis;
10. If there is evidence of a continuous discharge from the nose not caused by a sinusitis or antrum disease;
11. If the tonsil is "buried" and therefore objectively not visible as a large structure but shows definite evidence of a diseased condition.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

HOLLAND, CLYDE W. A.B., M.D. Dalhousie University Faculty of Medicine 1923. Rockefeller Fellow in Internal Medicine. Address: 119 Spring Garden Road, Halifax, Nova Scotia. Associated with him is:

LEVINE, SAMUEL A. A.B., M.D. Harvard University Medical School 1914. Assistant Professor of Medicine, Harvard Medical School. Senior Associate in Medicine, Peter Bent Brigham Hospital. Visiting Physician, Beth Israel Hospital. Address: 270 Commonwealth Avenue, Boston. Their subject is "Limitations of the Electrocardiogram as an Aid in the Diagnosis of Coronary Occlusion." Page 545.

KIEFER, EVERETT D. A.B., M.D. Harvard University Medical School 1926. Physician, Department of Gastroenterology, Lahey Clinic, Boston. His subject is "Chronic Ulcerative

Colitis." Page 552. Address: 605 Commonwealth Avenue, Boston.

DAYTON, NEIL A. M.D. Ohio State University College of Homeopathic Medicine, 1915. Formerly, Assistant Superintendent, Wrentham State School, Wrentham, Mass. At present Director of Research and Director of Division of Mental Deficiency, Massachusetts State Department of Mental Diseases. Member of the American Psychiatric Association, American Association for the Study of the Feebleminded, New England Psychiatric Society and American Statistical Association. Address: 109 State House, Boston. Associated with him are:

DOERING, CARL R. D.Sc., M.D. Baylor University College of Medicine 1921. Assistant Professor of Vital Statistics, Harvard Medical School. Address: Harvard Medical School, Boston. And

HILFERTY, MARGARET M. A.B. Technician, Harvard Medical School. Address: Harvard Medical School, Boston. And

MAHER, HELEN C. A.B. Technician, Harvard Medical School. Address: Harvard Medical School, Boston. And

DOLAN, HELEN H. S.B. Chief Research Worker, Department of Mental Diseases, State House, Boston. Address: Department of Mental Diseases, State House, Boston. Their subject is "Mortality and Expectation of Life in Mental Deficiency in Massachusetts: Analysis of the Fourteen-Year Period 1917-1930." Page 555.

WOODWARD, SAMUEL B. A.B., M.D. Harvard University Medical School 1878. His subject is "Arguments in Favor of Compulsory Vaccination for Private School Children." Page 570. Address: 58 Pearl Street, Worcester, Mass.

GREEN, ROBERT M. A.B., M.D. Harvard University Medical School 1906. Surgeon-in-Chief for Gynecology and Obstetrics, Boston City Hospital. Assistant Professor of Applied Anatomy, Harvard University. Instructor in Gynecology and Obstetrics, Harvard University. His subject is "History of the Massachusetts Medical Benevolent Society (A Personal Sketch)." Page 573. Address: 201 Bay State Road, Boston.

LEE, ROGER I. A.B., M.D. Harvard University Medical School 1905. His subject is "Massachusetts Medical Benevolent Society. Whom and How We Help." Page 574. Address: 264 Beacon Street, Boston.

PEMBERTON, FRANK A. S.B., M.D. Harvard University Medical School 1909. F.A.C.S. Instructor in Gynecology, Harvard University Medical School. First Assistant Visiting Surgeon, Free Hospital for Women. Member Surgical Staff of the New England Deaconess and Palmer Memorial Hospitals. His subject is "Progress in Gynecology." Page 575. Address: 198 Commonwealth Avenue, Boston.

MASSACHUSETTS LEGISLATIVE NOTES

THE CHIROPRACTIC HEARING

In addition to the speakers previously reported as having presented convincing arguments against the adoption of the chiropractic bill was Dr. Alexander S. Begg, whose address was one of the outstanding features of the hearing.

HOUSE—NO. 1214

Mr. Kendall of Dunstable presented a petition of James E. Kendall relative to appointment of health officers and financing of health districts by joint boards of health of two or more towns. Public Health.

THE COMMONWEALTH OF MASSACHUSETTS

In the Year One Thousand Nine Hundred and Thirty-Two

An Act relative to Appointment of Health Officers and Financing of Health Districts by Joint Boards of Health of Two or More Towns.

Be it enacted by the Senate and House of Representatives in General Court assembled, and by the authority of the same, as follows:

Section twenty-seven A of chapter one hundred and eleven of the General Laws is hereby amended by striking out said section twenty-seven A and inserting in place thereof the following:

SECTION 27A. The boards of health of two or more towns may, by vote of each, form a joint board of health for a district of said towns for the purpose of employing a health officer and such other assistants and clerks as may be determined by said joint board, who shall perform such duties and receive such compensation as said joint board shall prescribe. Said joint board shall appoint the treasurer of the town in the district where the main office of the joint board of health is located, or the treasurer of some other town in said district, or a member of said joint board, to act as treasurer of said district. The joint board shall estimate on or before January first of each year the cost of the district to be paid by each town and certify the same to each town, the same to be paid to said treasurer of said district.

The following bills have been given leave to withdraw:

HOUSE 1109—petition (from files of 1931) of Max Ulin for the establishment of the University of Massachusetts and to define its powers and duties.

SENATE 29 — petition of James G. Moran for legislation relative to the payment of reasonable charges of charitable hospitals in personal injury

cases under the compulsory motor vehicle insurance law.

HOUSE 1061 — petition of William Welch and others that employees injured in industrial accidents be not required to visit doctors designated by insurance companies or the Department of Industrial Accidents.

HOUSE 929 — petition (from files of 1931) of Joseph A. Conway for the establishment of the Massachusetts Medical Research Board for the purpose of studying and distributing certain facts and information.

MISCELLANY

MORTALITY RATES

Telegraphic returns from 83 cities with a total population of thirty-six million for the week ending March 5, indicate a mortality rate of 13.4 as against a rate of 13.8 for the corresponding week of last year. The highest rate (25.5) appears for Wilmington, Del., and the lowest (5.9) for Yonkers, N. Y. The highest infant mortality rate (15.4) appears for Indianapolis, Ind., and the lowest for New Haven, Conn., Paterson, N. J., St. Paul, Minn., and Tacoma, Wash., which reported no infant mortality.

The annual rate for 83 cities is 12.3 for the nine weeks of 1932, as against a rate of 14.3 for the corresponding period of the previous year.

DR. LONG SUCCEEDS DR. OPIE

Dr. Esmond R. Long, Professor of Pathology at the University of Chicago, has been elected to the chair of pathology at the University of Pennsylvania School of Medicine and to the directorship of the laboratory in the Henry Phipps Institute for the Prevention and Cure of Tuberculosis. He will begin his duties on July 1, 1932, succeeding Dr. Eugene L. Opie who will become head of the Department of Pathology of Cornell University Medical School.—*Bulletin of the National Tuberculosis Association.*

HARVARD MEDICAL SCHOOL

Scholarship awards to First Year students for 1931-32:

Name of Scholarship—Awarded to:
David Williams Cheever—John R. Mote.
Charlotte Greene—Dale G. Friend.
George Haven—Leslie M. Bell.
George Haven—Albert W. Cowan.
George Haven—Edmund J. Croce.
George Haven—Lewis A. Giffin.
George Haven—Monroe T. Gilmour.
George Haven—John H. Grindlay.
George Haven—Donald T. Hall.
George Haven—Gilbert M. Jorgensen.
George Haven—Charles D. Roberts.
George Haven—James E. Robertson.
George Haven—Douglas A. Sunderland.
George Haven—William L. Wallbank.
George Haven—John H. Warner, Jr.
George Haven—Francis J. West.

Clark Wright Heath, M.D., 1926 (Harvard) has been awarded a William O. Moseley, Jr., Travelling Fellowship for 1932-33.

The following resignations have been received to take effect September 1, 1932:

Frank Burr Mallory, M.D., S.D., as Professor of Pathology (becoming Emeritus).

William Henry Robey, M.D., as Clinical Professor of Medicine (becoming Emeritus).

CORRESPONDENCE

THE RESTORATION OF DR. I. F. ARMSTRONG'S LICENSE TO PRACTICE IN MASSACHUSETTS

The Commonwealth of Massachusetts
Department of
Civil Service and Registration
Board of Registration in Medicine
State House, Boston

March 8, 1932.

New England Journal of Medicine,

This is to notify you that the license of Dr. Irving F. Armstrong, of Hudson, Massachusetts, to practice medicine in this Commonwealth was restored to him on March 3, 1932.

Yours truly,
STEPHEN RUSHMORE, M.D., *Secretary.*

A LETTER TO HON. R. S. COPELAND RESPECTING A PROPOSED CHANGE IN BANKRUPTCY

March 4, 1932.

Hon. Royal S. Copeland, M.D.,
The Capitol,
Washington, D. C.

My dear Senator Copeland,

As a member of the medical profession, whose pastime for many years has been the study of medical ethics and economics as they relate to a changing economic world, I am naturally alert to any proposed legislation which may affect the profession adversely.

At the present time, and in the past, the medical profession has protected itself against malpractice suits by the slender reed of liability insurance. Signs are not wanting that even this support may be withdrawn from it. It is common talk, that the insurance interests are not keen for this particular business. This, of course, is due to the greatly increased amount of litigation of this character. Should this protection fail, I feel that ninety-nine per cent of the medical profession would have to interest itself in the status of the bankruptcy law. A business wrecked by an adverse malpractice verdict against a physician, does not fall into the category of an ordinary business failure, but may be classified as a calamity, akin to an "Act of God."

With this preamble, Doctor, I call your attention

to President Hoover's proposed changes in the bankruptcy law.

With the President's evident desire to eliminate fraudulent bankruptcies I have no quarrel. In fact, I think that he is acting for what he believes to be the greatest good of the majority. I believe that the proper time to point out possible injustices in proposed legislation is before and not after passage. I suggest that the medical malpractice verdicts be exempt from the President's proposed changes, in so far as they relate to his suggestion, that bankrupts may be subject to confiscation in whole or in part, of "after acquired" earnings.

It seems to me that plaintiffs in these cases should be compelled to stand or fall on the physician's financial status at the time of the verdict, and that under no circumstances should they be allowed to take back a mortgage on his future earnings.

Any other construction of this matter would be to subject the medical profession to a system of peonage. It would make cowards of physicians, whose every act requires a certain amount of daring.

I am mailing you this letter, Doctor, a copy of which I am sending to the *New England Journal of Medicine*, and it is not to be construed in any sense as a criticism of the President's initiative. It has, rather, for its purpose, the pointing out of the fact that for every good there may be a little evil.

With best wishes I remain,

Very truly yours,

JOHN J. HURLEY, M.D.

EXTRACT FROM A LETTER REFERRING TO THE STATE BOARD OF HEALTH BY A NINETEENTH CENTURY PHYSICIAN

Boston, Thursday 3d December 1874

My dear ——,

I enclose an article from the *Journal* of this morning that I have read with interest and think it will be additionally interesting to you—also an article of Sanitary import from the same:—date of Nov. 10.— Have you ever given any attention to the reports of our "State Board of Health" now having existed about six years? The information contained in these reports is at the expense of the state—gathered from all sources and relating to all sanitary questions affecting the public health—Air—Light—Ventilation—Dress—Diet—Location — Drainage—Poisons—Adulterations of food—Occupations, etc. The matter has long been one of deep interest to me and put as these investigations—reports and criticisms are in simple and easily comprehended terms, divested of all professional terms and forms it becomes as entertaining as a romance almost and if extensively circulated as the object is, must result in great good in instructing the people concerning these principles so vital to, and underlying all human life.

INSTRUCTION IN HEART DISEASE

Instruction Letter No. 23.
March 3, 1932.

To School Physicians:

I am pleased to present the following statement relative to the diagnosis of heart disease in childhood written by Dr. Paul Dudley White, Physician, Massachusetts General Hospital, and Instructor at Harvard University Medical School. School physicians are requested to give this matter their earnest attention.

Very truly yours,

SOLOMON H. RUBIN, M.D.,
Acting Director of School Hygiene.

CRITERIA FOR THE DIAGNOSIS OF HEART DISEASE IN CHILDHOOD

A. POSITIVE EVIDENCE

1. *Enlargement.* The apex impulse and left border of dullness to percussion in the fifth intercostal space must be to the left of the midclavicular line (which is the vertical line drawn midway between the midsternal line and the outer end of the clavicle). Two qualifications should be added: (a) displacement of the heart to the left must be carefully distinguished from enlargement (such displacement results from pressure exerted by conditions in the right thoracic cavity or from retraction of the left lung and pleura); (b) in infants or in fat, short children with high diaphragms the apex impulse and left border of dullness may be found slightly outside the midclavicular line (up to 1 centimeter or 1/2 inch) if in the 4th intercostal space.
2. *Diastolic or continuous murmurs* heard anywhere over the heart, provided they are unquestionable and provided a continuous murmur at the base of the heart is not simply transmitted there from a venous hum at the base of the neck. Such a venous hum is a normal physiological occurrence best heard upright; it is easily blocked by pressure on the neck veins.
3. A palpable thrill over the heart, whether it be systolic, diastolic, or continuous in time, provided it is unquestionable.
4. Cyanosis of the lips and clubbing of the fingers in the absence of chronic pulmonary disease.
5. Clear x-ray evidence of enlargement or abnormal shape, or both. Often heart disease has been diagnosed wrongly on insufficient x-ray evidence.
6. Clear abnormality of the electrocardiogram, particularly auricular fibrillation, heart-block, and marked axis deviation. Sinus arrhythmia, premature beats, and paroxysmal tachycardia do not mean that heart disease

is present. It must be appreciated that there are wide limits of normal in electrocardiography.

B. Strongly Suggestive Evidence

1. Bulging of the chest over the precordium.
2. Very loud harsh systolic murmurs anywhere over the precordium, not made to disappear on full inspiration.
3. Dyspnea unexplained by pulmonary disease or neurocirculatory asthenia.
4. Engorgement of the neck veins with enlargement of the liver, which may occur with either congestive heart failure, marked mitral stenosis, pericardial effusions, or chronic constrictive pericarditis (Pick's disease).
5. Clear history of rheumatic fever or chorea.

C. Somewhat Suggestive Evidence

1. Very loud apex first sound.
2. Very loud pulmonary second sound.
3. Moderately loud systolic murmur, especially at the apex.

NOTICES

Robert L. Mason, M.D., has opened an office at 475 Commonwealth Avenue, Boston.

TRAVEL TOURS FOR 1932

Literature, designed to secure bookings for the Coöperative Clinic Tours in connection with the Hundredth Anniversary Meeting of the British Medical Association, has been disseminated by the Amerop Travel Service. The *Journal* will be pleased to furnish further information to anyone who contemplates a trip abroad this summer.

RADIO HEALTH MESSAGES

March, 1932

Sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

Courtesy WBZA. Tuesdays at 5:00 P. M.

March

22 Can High Blood Pressure Be Avoided?

29 Rose Cold and Hay Fever

State House Broadcast

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays at 12:30 P. M.

Health matters of public interest discussed by the Division Directors of the Department.

Radio Health Forum

Queries from the public are answered under the sponsorship of the Department of Public Health.

Courtesy WEEI. Fridays at 4:50 P. M.

Questions on Health and Prevention of Disease may be sent to Radio Health Forum, State Department of Public Health, State House, Boston.

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P. M. on Thursday, March 24, in the Amphitheatre of the Peter Bent Brigham Hospital, a medical clinic will be given by Dr. Henry A. Christian, to which physicians are cordially invited. The clinics will be held regularly on Thursday afternoons.

REPORTS AND NOTICES OF OF MEETINGS

THE HARVARD MEDICAL SOCIETY

A regular meeting of the Harvard Medical Society was held in the amphitheatre of the Peter Bent Brigham Hospital on March 8, 1932. Dr. Harvey Cushing presided.

A medical case was presented by Dr. Robert Parsons. A sixty-two year old man was admitted to the hospital because of precordial pain. Three years previously he began to have attacks of epigastric pain which radiated toward the region of the heart and were usually relieved by belching. Ten days before entry he was seized by a sudden severe precordial pain which radiated down the left arm and lasted throughout the night. A second attack on the following evening was relieved by a hypodermic injection. Similar attacks occurred each night before entry. Physical examination showed that the heart was moderately enlarged to the left. The blood pressure was 210/124 on entry, 128/84 a week later, and 190/100 two weeks later. During this time he had frequent nocturnal attacks of severe anginal pain usually relieved by nitroglycerin although morphine was required on one occasion. Because of the frequency and severity of the pain it was considered advisable to inject the left sympathetic chain with alcohol. This was done by Dr. John Homans on February 24 and the patient has had no anginal pain since that date. On the evening of the injection, signs of a partial pneumothorax were noted on the left and the diagnosis confirmed by x-ray. Presumably, in a tentative misdirection of one of the needles — afterward corrected — the lung was pierced, allowing air to escape into the pleural cavity. It is unlikely that any alcohol entered the cavity. Such a complication of paravertebral sympathetic injection had not previously been seen at the Peter Bent Brigham Hospital. Since the injection he has had some intercostal neuralgia due, undoubtedly, to an alcoholic neuritis of the intercostal nerves. This complication is frequently seen but the neuralgic pain usually disappears after some time and leaves the patient free from all pain.

Dr. David Gaiser presented a surgical case. A thirty-five year old man began to have a burning

epigastric pain seven years ago. It usually came on an hour after meals and was relieved by soda. At times there were nausea and vomiting. Following treatment with a milk and cracker diet four years ago, he was free from all symptoms for nine months. Since then there has been a number of remissions and exacerbations. During the nine months prior to entry the pain has been more severe than before and the usual remedies have given no relief. There was no history of hematemesis or melena. Roentgen examination showed a hypomotile stomach, a filling defect on the lesser curvature near the incisura, and an irregular constriction of the pylorus with twenty-five per cent retention at six hours. He was placed on a strict Sippy régime with nightly aspirations. After a month he was discharged free from all symptoms. X-ray at the time of discharge showed almost complete disappearance of the ulcer but the constriction of the pylorus was slightly increased. Two weeks later symptoms returned and he was readmitted. X-ray showed that the ulcer had entirely disappeared but there was an annular constriction of the pylorus suggesting malignancy. An exploratory laparotomy was done and not only was no evidence of malignancy found but the pyloric region was hardly thickened beyond normal limits. Because obstruction had progressed in spite of thorough medical treatment, the pyloric end of the stomach was resected and the stomach anastomosed to the duodenum. Pathologic examination showed two minute healing ulcers of the pylorus and no gross evidence of malignancy. The postoperative course has been uneventful.

Dr. Francis G. Benedict, Director, Carnegie Nutrition Laboratory, spoke on "Giant Tortoises and Pythons and their Contribution to Physiology." For many years Dr. Benedict and his associates have been studying metabolism and heat regulation of warm and cold-blooded animals. Giant tortoises and snakes have been used for the latter studies because they are comparable in size to man. Almost any change in the body temperature of a cold-blooded animal may be produced simply by changing the temperature of the environment. The skin temperature of the python or giant tortoise is always just slightly below that of the environment. The cloacal and mouth temperatures are equal and slightly below that of the skin. Hence the animal is constantly in a position to absorb heat from its surroundings rather than to give off heat as does the warm-blooded animal. The ability of cold-blooded animals to absorb heat from the environment was conclusively proved by calorimetric experiments.

As it is possible to vary the body temperature of the python as much as twenty or thirty degrees it furnishes an excellent animal in which to study the temperature coefficient of metabolism. The law of van't Hoff states that the velocity of a chemical reaction is doubled for each rise in temperature of ten degrees centigrade. Du Bois, studying the heat production in fever, concluded that the law held for man in the fever range. Dr. Benedict studied this coefficient in the python and found that for each ten degrees rise in temperature the total metabolism is increased two and one-half times.

The total heat production of the cold-blooded animal is much less than of the warm-blooded animal. Dr. Benedict has raised the body temperature of pythons to 37° C. and of rattlesnakes to 44°C. Even at these high temperatures the heat production of a cold-blooded animal is only about one-eighth that of a warm-blooded animal. Such a relation might be expected in view of the creature's small heart and very scanty body fluids.

In conclusion, Dr. Benedict stated that experimental studies with giant tortoises and pythons had yielded little information of direct importance to human physiology. Nevertheless it has added to the knowledge of comparative physiology and eventually may be of practical importance.

THE ESSEX SOUTH DISTRICT MEDICAL SOCIETY

The Essex South District Medical Society held its regular meeting at Lynn Hospital on March 2, 1932, at five o'clock P. M.

The following clinical program was presented:

1. Gangrene of the Lung. Stephen R. Davis, M.D.
2. A New Thought on Treatment of Hemophilia. Albert H. Covner, M.D.
3. a. Bone Lesion of the Femur?
   b. End Results in Case of Multiple Fracture.
   c. Gas Bacillus Lower Extremity. Charles Damsky, M.D.
4. a. Renal Tuberculosis.
   b. Pyonephrosis. Edward L. Peirson, Jr., M.D.
5. Placenta Accreta. Richard J. Williams, M.D.
6. Solitary Abscess of the Liver. Earl Newman, M.D.
7. Friedman Test for Pregnancy. Thomas E. Culliton, M.D.
8. Cranio-Cerebral Injuries. William T. Hopkins, M.D.

Following dinner in the nurses' dining room the Society was addressed by Dr. Elmer W. Barron of Boston, Professor of Pediatrics at Tufts Medical School and Chief of the Floating Hospital. His subject was Infant Foods and Infant Feeding.

The evening meeting was held in Ryan Hall in the new home for nurses.

WM. T. HOPKINS, *Reporter.*

SOUTHBRIDGE DISTRICT MEDICAL SOCIETY

The Southbridge District Medical Society entertained the members of the Wachusett Medical Society, Webster Medical Society, and a few invited guests of the Worcester County District Medical Society at the monthly meeting held Thursday, March 3, at the Cohasset Country Club. The guest speaker of the evening was Dr. Howard Clute of the Lahey Clinic, who spoke on diseases of the gall bladder, with illustrations.

THEODORE L. STORY, *Secretary.*

NATIONAL TUBERCULOSIS ASSOCIATION

TWENTY-EIGHTH ANNUAL MEETING—
TENTATIVE CALENDAR

Colorado Springs
June 6, 7, 8 and 9, 1932

Meetings to be held in The Antlers Hotel

MONDAY, JUNE 6

10:00 A. M.—American Sanatorium Association.
National Conference of Tuberculosis Secretaries.
12:30 P. M.—Christmas Seal luncheon.
2:00 P. M.—American Sanatorium Association.
National Conference of Tuberculosis Secretaries.
4:00 P. M.—Board of Directors, National Tuberculosis Association.
8:15 P. M.—Opening General Meeting—National Tuberculosis Association.
President's Address.
Report of Managing Director.
Award of Trudeau Medal.
Report of Committee on Nominations.

TUESDAY, JUNE 7

9:30 A. M.—Clinical Section.
Sociological Section.
12:30 P. M.—Luncheon, Rocky Mountain Tuberculosis Conference.
2:00 P. M.—Pathological Section.
Administrative Section.
6:30 P. M.—National Tuberculosis Association Dinner.
10:00 P. M.—Dancing.

WEDNESDAY, JUNE 8

9:30 A. M.—Pathological Section.
Administrative Section.
12:30 P. M.—Luncheon Meetings.
2:00 P. M.—Clinical and Pathological Sections (Joint Session).
Sociological Section.
4:30 P. M.—Board of Directors Meeting.
8:00 P. M.—X-ray Conference.

THURSDAY, JUNE 9

9:30 A. M.—Clinical Section.
Sociological Section.
12:15 P. M.—General Meeting.
2:00 P. M.—Pathological Section.
Administrative Section.

For particulars address the Executive Secretary, Mr. Frederick D. Hopkins, 450 7th Ave., New York City.

BOSTON MEDICAL HISTORY CLUB

Sprague Hall, 8 The Fenway, Boston
Monday, March 21, 1932, at 8:15 P. M.

Ramazzini. Father of industrial medicine. Francis D. Donoghue, M.D. Member Permanent International Committee on Industrial Accidents. Medical Advisor Industrial Accident Board of Massachusetts.

The Ancient and Modern Practice of Medicine in China. Medical experiences in City and Country. Theodore C. Greene, M.D., of Peiping, China. Illustrated with colored stereopticon slides.

Physicians and medical students and their friends cordially invited.

JAMES F. BALLARD, *Secretary.*

SUFFOLK DISTRICT MEDICAL SOCIETY

The March meeting of the Suffolk District Medical Society will be a clinical meeting to be held at the New England Deaconess Hospital, 16 Deaconess Road, Boston, on Wednesday, March 30, at 8:15 P. M., promptly.

CLINICAL PROGRAM

1. Four Interesting Foreign Bodies in the Upper Air and Food Passages. Dr. Lyman Richards.
2. New Methods in the Practical Treatment of Diabetes. Dr. E. P. Joslin.
3. The Increasing Incidence of Pernicious Anemia in Diabetes Mellitus and Their Relation to One Another. Dr. Howard F. Root.
4. Presentation of Cases. Dr. F. Gorham Brigham.
5. Carcinoma of the Prostate. Dr. G. G. Smith.
6. Hyperthyroidism and Heart Failure. Dr. H. M. Clute.
7. Cancer of the Colon and Rectum. Dr. R. B. Cattell.
8. Differentiation of Gastric Ulcer and Gastric Cancer. Dr. Sara M. Jordan.

REGINALD FITZ, M.D., *Vice-President.*
LELAND S. MCKITTRICK, M.D., *Secretary.*

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance), Tuesday evening, March 22, at 8:15 o'clock.

PROGRAM

Presentation of Cases.

Calcification of Arteries and the Associated Osteitis Fibrosa, by Dr. W. G. MacCallum, Professor of Pathology, Johns Hopkins Medical School.

JOHN HOMANS, M.D., *Secretary.*

BOSTON CITY HOSPITAL

The next Staff Clinical Meeting will be held Thursday, March 24, 1932, at 8:15 P.M., in the Cheever Amphitheatre.

Dr. James J. Hepburn.
Cholelithiasis.

Dr. George W. Papen.
1. Duodenal Fistulae.
2. Secondary Vesical Calculus.

Dr. Horace Binney.
1. Cancer of Gall Bladder.
2. Pulmonary Embolectomy.

Dr. Newton C. Browder.

Points in Connection with Treatment of Colles Fracture.

Dr. Lewis R. Dretler.

A Case of Traumatic Pressure Pneumothorax.

Doctors and medical students cordially invited.

COMMITTEE ON HOSPITAL CLINICS.

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

March 17—New England Hospital for Women and Children. See page 542, issue of March 10.

March 17—New England Physical Therapy Society. See page 541, issue of March 10.

March 18—The New England Roentgen Ray Society. See page 541, issue of March 10.

March 17 - April 28—Harvard Graduate School of Medicine. Worcester Extension Course. See page 537, issue of March 10.

March 18—A Lecture on Clinical and Anatomic Findings in Fulminating Rheumatism. See page 537, issue of March 10.

March 21—Boston Medical History Club. See page 598.

March 22—Harvard Medical Society. See page 598.

March 24—Medical Clinic at the Peter Bent Brigham Hospital. See page 596.

March 24—Boston City Hospital. See page 598.

March 26—Examination Conducted by the American Board of Obstetrics and Gynecology (written) — May 10 (general, oral and clinical). See page 252, issue of February 4.

March - April, Autumn — International Postgraduate Courses in Berlin. See page 1169, issue of December 10.

April 4-8—The American College of Physicians. See notice on page 1232, issue of June 4.

April 6 - June 1—New England Heart Association. See page 891, issue of October 29.

April 7 and 8—Clinics for the Demonstration of Cured Cancer Cases. See page 486, issue of March 3.

May 9-13—American Medical Association, New Orleans, La.

June 6, 7, 8, and 9—National Tuberculosis Association. See page 598.

June 8, 9, 10—Massachusetts Medical Society, Hotel Statler, Boston.

June 21-24—Catholic Hospital Association. See page 369, issue of February 18.

September 19-22 — International Congress of Biliary Lithiasis. See notice on page 427, issue of February 25.

October 24-27 — American Public Health Association. See notice on page 1321, issue of December 31.

October 24-28—Inter-state Postgraduate Medical Association of North America. See page 39, issue of January 7.

DISTRICT MEDICAL SOCIETIES

Essex North District Medical Society

May 4—Annual meeting at Lawrence.

Essex South District Medical Society

April 6, Wednesday—Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speaker, Dr. Arthur P. Wakefield, State Department of Health. Subject: 10-Year Tuberculosis Program in the Public Schools.

May 5, Thursday—Censors' Meeting, Salem Hospital, 3:30 P.M.

May 10, Tuesday—Annual Meeting. The Tavern, Gloucester. Speaker and subject to be announced later. Ladies invited.

Franklin District Medical Society

May 10—Meeting at the Weldon, Greenfield, at 11 A.M.

CHARLES MOLINE, Secretary.

Middlesex East District Medical Society

May 11 (Unless otherwise announced)—At Woburn.

Norfolk District Medical Society

March 29—Place to be announced. Boston Public Health Meeting. Speakers, Honorable James M. Curley, Dr. Francis X. Mahoney, Dr. Charles Wilinsky, Dr. W. H. Griffin.

May—Annual Meeting, date and place to be announced.

May 5—Censors' Meeting for examination of candidates. Roxbury Masonic Temple, 171 Warren Street, Roxbury, 4 P.M. Fee $10.00 payable at time of examination. All candidates will have applications in the hands of the secretary one week prior to this date. All candidates from Medical Schools not on approved list of Society must have application complete two weeks prior to date of examination.

F. S. CRUICKSHANK, Secretary.

Norfolk South District Medical Society

April 7—To be announced later.

May 5—Annual Meeting.

The time of all meetings is 12 o'clock noon, at the Norfolk County Hospital, South Braintree. Censors will meet on May 5 at 11:15 A.M. at the Norfolk County Hospital.

N. R. PILLSBURY, Secretary.

Suffolk District Medical Society

March 30—See page 598.

April 27—The Annual Meeting, Boston Medical Library. Election of Officers.

The Medical Profession is cordially invited to attend these meetings.

REGINALD FITZ, M.D., Vice President.
LELAND S. McKITTRICK, M.D., Secretary.
HILBERT F. DAY, M.D., Boston Medical Library.

Worcester District Medical Society

Wednesday evening, April 13—Grafton State Hospital, North Grafton, Mass. 6:30—Dinner, complimentary by Grafton State Hospital. 7:45—Program to be announced later.

Wednesday afternoon and evening, May 11—Annual Meeting. Program to be announced later.

ERWIN C. MILLER, Secretary.

BOOK REVIEWS

Surgical Pathology of the Female Generative Organs. By ARTHUR E. HERTZLER, M.D. J. B. Lippincott Co. Philadelphia, 1932. Pp. 346. Price $5.00.

This volume represents one of a series on surgical pathology prepared by Dr. Hertzler. It is essentially a slightly expanded catalogue of the disease processes which involve the female genitalia. Occasionally there is some brief discussion. One cannot but feel that this book has fallen more than a little short of what it might have been. It is profusely illustrated and in the gross photographs lies the chief value of the work.

At best the discussions of the various lesions are sketchy and far from fully informative. If the trained gynecologist had need for a quiz compend in his specialty this book might serve excellently well. Minor inaccuracies, together with scattered infelicities of expression, somewhat mar the text.

In view of the general acceptance of Dr. Sampson's work it is rather surprising on page 67 to note under adenomyoma of the tube "Nothing is known of their genesis but from their structure it is likely that they are congenital in origin." In the same way, in discussing adenomyoma of the uterus Sampson's work is entirely disregarded.

Judging from pages, such as the following, Dr. Hertzler's experiences with pathologists have been unfortunate. On page 135 we find "Fortunately for the pathologist surgeons are credulous. . . . Pleasant practical relations with the pathologist and not sci-

entific accuracy is the dominant factor in the average operator's mind. . . . Man to man this striving for harmony is very commendable but it leads to the removal of organs that have never threatened the life of the patient." Also the dictum on page 135, "All sections removed for diagnostic purposes should be removed by the pathologist," is neither practical nor advisable.

One fine section is the discussion on needless oöphorectomy. Anything which will retard the all too enthusiastic rush for excision of ovaries with dilated follicles or with corpora is to be welcomed.

Radium is hardly mentioned in this volume. The attitude of the author is perhaps best expressed on page 178 in a discussion of carcinoma of the cervix: "That the outlook is gloomy is evidenced by the fact that the majority of experienced surgeons are quite willing to relegate the management of this disease to the roentgenologists." Then on the same subject (page 179) we read: "However, reported cases are accompanied by so little real evidence that it is impossible to compare results. Merely publishing a photomicrograph presenting a high magnification is insufficient."

While this book should do more good than harm, it is a pity it could not have been a little better.

Monographs of the Pickett-Thomson Research Laboratory. Volume 1. *The Vitamins.* By ETHEL BROWNING, M.D. Williams and Wilkins Co., Baltimore, 1931. Pp. 575. Price $10.00.

The opening paragraph of the author's introduction states, "The original purpose of this monograph was to collect into one volume as much as possible of the existing literature on the subject of Vitamins, and to present to the scientific reader a comprehensive account of the researches which have established the vitamin theory on its present basis. That purpose has been followed as strictly as limitations of time and space would allow, but the literature on the subject has assumed such colossal proportions, while opinions, even amongst those who have devoted years of study to only one aspect of the vitamin problem have proved so divergent, that the author has had perforce to remain content with the endeavor to weave into a complete pattern those threads which have seemed most important to the continuity of the design."

This avowed purpose has been achieved in truly remarkable fashion. A wealth of material is presented in orderly arrangement. Following a consideration of vitamins in general, the three fat-soluble vitamins, A, D and E, and the two water-soluble vitamins, B and C, are taken up separately. Extensive tables showing the vitamin content of foodstuffs are followed by an equally complete bibliography and index. One would hardly recommend the monograph to those interested in the vitamins superficially; but it should prove to be of inestimable value as a book of reference for workers on problems concerning one or another of the aspects of this important subject.

Studies from the Rockefeller Institute for Medical Research. Reprints. Volume 79. New York. 1931. Pp. 626.

This volume, representing the recent work of the Rockefeller Institute and Hospital, contains a rather larger proportion of papers in the field of chemistry and the basic medical sciences than usual. An interesting study of psittacosis is presented by Rivers and his coworkers.

A curious organism isolated from marsh peat which dissolves the capsule of Type III pneumococcus is described, together with its manner of action, by Avery and Dubos.

The remainder of the papers have but little interest from the clinical standpoint with the exception of a discussion of the onset of rheumatic fever by Swift.

BOOKS RECEIVED FOR REVIEW

Clinical Roentgen Pathology of Thoracic Lesions by William H. Meyer. Published by Lea & Febiger. 272 Pages. Price $6.00.

Cancer—What Everybody Should Know About It by James A. Tobey. Published by Alfred A. Knopf. 313 Pages. Price $3.00.

The Practice of Contraception. Edited by Margaret Sanger and Hannah M. Stone. Published by The Williams and Wilkins Company. 316 Pages. Price $4.00.

Prohibiting Minds and The Present Social and Economic Crisis by Stewart Paton. Published by Paul B. Hoeber, Inc. 198 Pages. Price $2.00.

Mental Healers — Franz Anton Mesmer. Mary Baker Eddy and Sigmund Freud by Stefan Zweig. Translated by Eden and Cedar Paul. Published by The Viking Press. 363 Pages. Price $3.50.

A Hand-book of Ocular Therapeutics by Sanford R. Gifford. Published by Lea & Febiger. 272 Pages. Price $3.25.

The Surgical Clinics of North America. February, 1932, Volume 12, Number 1. Chicago Number. Published by W. B. Saunders Company. 240 Pages.

Surgery of The Chest by George F. Straub. Published by Charles C. Thomas. 475 Pages. Price $10.50.

United States Army X-Ray Manual. Second Edition, Rewritten and Edited by Lt. Col. H. C. Pillsbury. Published by Paul B. Hoeber, Inc. 482 Pages. Price $5.00.

Applied Physiology by Samson Wright. Fourth Edition. Published by Humphrey Milford, Oxford University Press. 552 Pages. Price $5.50.

Thomson & Miles' Manual of Surgery by Alexander Miles and D. P. D. Wilkie. Volume Second and Third. Eighth Edition. Volume 2, 685 Pages. Volume 3, 578 Pages. Price $3.80 each.

The Wisdom of the Body by Walter B. Cannon. Published by W. W. Norton & Company, Inc. 312 Pages.

Studies from the Rockefeller Institute for Medical Research. Reprints, Volume 80. Published by The Rockefeller Institute for Medical Research. 637 Pages.